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CONTENTS OF PREVIOUS NUMBER

OCTOBER, 1927. NUMBER 4

The Corpus Striatum: A Study of Secondary Degenerations Following Lesions in Man and of Symptoms and Acute Degenerations Following Experimental Lesions in Cats. Lawrence Onis Morgan, Ph.D., Chicago.

Intra-Ocular and Intracranial Pressure: An Experimental Study. Frank Fremont-Smith, M.D., and Henry S. Forbes, M.D., Boston.

The Acetic Anhydride-Sulphuric Acid Test: Further Data on the Boltz Cerebrospinal Fluid Test. Lee D. Cady, M.D., St. Louis.

Clinical and Occasional Notes:

Progressive Neuritic Muscular Atrophy. A. E. Kulkov, M.D., Moscow, U. S. S. R.

Adenoma of the Hypophysis Without Acromegaly, Hypopituitarism or Visual Disturbances, Terminating in Sudden Death. Esmond R. Long, M.D., Chicago.

Psychosis in a Child Aged Seven Years. Henry H. Hart, M.D., Greenwich, Conn.

The Neuropathic Taint in Migraine. William Allan, M.D., Charlotte, N. C.

Hexyiresorcinol as a Cerebrospinal Antiseptic. H. P. Schenck, M.D., Philadelphia.

News and Comment:

Postgraduate Course in Neuropsychiatry.

New Journal of Psychoanalysis.

Abstracts from Current Literature.

Society Transactions:

Philadelphia Neurological Society.

New York Neurological Society.

Société de Neurologie de Paris.

Chicago Neurological Society.

Book Reviews.

Archives of Neurology and Psychiatry

VOLUME 18 NOVEMBER, 1927 NUMBER 5

STUDIES IN STUTTERING

INTRODUCTION

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IOWA CITY

In the course of a study of certain cases of strephosymbolia [1] in 1925, I noted that among my first series of fifteen cases there were three patients who stuttered or who had formerly done so and four others whose speech had a peculiar laboring hesitancy like that of one who has been broken of stuttering. There are a number of instances recorded in the literature of the onset of stuttering when a normally left handed child is coerced into using the right hand for writing and of recovery when the use of the left hand is permitted. One such case of my own series is striking:

A college student, aged 18, who was naturally left handed and who had been permitted to use the left hand in writing during his first two years in school, was required by the teacher in the third grade to shift to the right hand; this shift was carried over to the use of knife and fork and to other activities. In the following year he began to stutter, and he was a moderately severe stutterer from that time until his freshman year in college, when he was referred to the Psychopathic Hospital by the Department of Speech of the State University of Iowa. He considered himself right handed at this time, but when he was tested in writing with either hand in both dextral and sinistral directions, i. e., both normal and mirrored writing, it was found that even after nine years of practice with the right hand he had a markedly greater facility with the left. The letters were better formed and more rapidly made; he made fewer errors in spelling and the product was better in content and construction. Experiments in simultaneous blackboard writing with both hands, together with the results of other examinations, led me to feel that the left was a much more facile avenue of motor function for him than the right, and he was advised to make an effort to use the left hand for everything.

This experiment was begun in October, and the parents reported a marked betterment in speech when he went home for the Christmas vacation. From then on he showed rapid improvement, and within seven months after the shift the stuttering had entirely disappeared. Indeed, by the end of the scholastic year his classmates were becoming amused at his volubility; he was making up lost time, so to speak, after many years of handicap.

Another of the patients is really a severe stutterer who apparently also should use the left hand. I have tried the experiment of making

1. Orton, S. T.: "Word-Blindness" in School Children. Arch. Neurol. & Psychiat. **14**:581 (Nov.) 1925.

him write with the left hand while he is attempting to speak, with the aim of thus determining a consistent right cerebral lead. During this action he can talk with comparative freedom, though slowly, of course, in order not to get ahead of his pencil. Without this guide, he has the greatest difficulty.

These and other suggestive cases seem to support the theorem that stuttering, like the reading disability—strephosymbolia—is often an expression of confusion in cerebral dominance. The larynx and other organs of speech, unlike the limbs, are not independent, paired organs, but are single mechanisms, though activated by paired groups of muscles on either side of the midline. The possibilities for great difficulty here through confusion in dominance or through alternating dominance are obvious. Under this conception, stuttering would be more closely related to the apraxias than to the ataxias. The act of stuttering is not unlike an ataxia, and this has led to the suggestion that the cerebellum may be at fault. One does not, however, observe stuttering as a result of demonstrable cerebellar lesions and there are, moreover, reasons to believe that speech and writing are both essentially integrative functions of the higher cortical arcs of the dominant hemisphere.

The emotional variants to be discerned in many stutterers have also been held as explanatory of the difficulty. Particularly is this emphasized in those cases in which the stuttering seems most apparent in strange or embarrassing company. It is often, however, just here that the speech partakes most strikingly of the propositional. Unquestionably such a disability would react on the emotions and give rise to a definite reaction pattern unlike that of the normal speaker; indeed, in the cases of reading disability in children of the lower school grades, it is felt that steps in the genesis of emotional disturbances developing from this condition rather than determining it have been observed.

In June, 1925, a grant was made by the Rockefeller Foundation to the Iowa Psychopathic Hospital for a two-year program of research in physiology of the brain, with these studies in cerebral dominance as its central point. Work on this program was started in January, 1926, and the work with stutterers was entrusted to L. E. Travis, who was already at work in the hospital laboratories on similar lines under a Fellowship of the National Research Council. The first of Travis' studies appears in this issue of the ARCHIVES and seems to bear out the idea of a marked lack of integrative solidarity—a dysintegration—in the stutterer's attempts at speech. Further work of a suggestive nature is under way.

STUDIES IN STUTTERING*

I. DYSINTEGRATION OF THE BREATHING MOVEMENTS DURING STUTTERING

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The complex peripheral structures serving speech may be divided into three main functional groups: those of breathing, voice and articulation. It is possible to study the muscular movements within each of these groups and the various possible combinations of movements between them. Normal speech displays exquisite harmony within the separate groups and in their combinations, while certain types of abnormal speech, notably stuttering, show a disharmony in the combination of the three primary groups of muscles and also within each group.

Halle,[1] Ten Cate,[2] Gutzmann,[3] Fletcher[4] and others have studied spasms in the breathing musculature of the stutterer, and all agree that tonic and clonic spasms occur in all cases. Halle and Ten Cate were among the earliest investigators in this field, and both recorded various disturbances and abnormalities in breathing. Fletcher made an intensive study of the temporal relationships between thoracic and abdominal breathing. Gutzmann gave an excellent analysis of the breathing, laryngographic and lip movements in typical cases and attempted to determine what part of the speech mechanism gave the first indication of disturbance.

The present paper is a report on some studies of the faulty integration of the major muscular units of the breathing function during stuttering. It concerned itself chiefly with movements of the abdomen and thorax, with changes in pressure of the breath stream and with the vertical movements of the larynx. It does not include studies of articulation or of voice. Both of these groups are under investigation, and a report of one of them forms the second paper of this series.

* The work here recorded was carried out in the laboratories of the Iowa State Psychopathic Hospital as part of the program of research in physiology of the brain which is being supported by a grant from the Rockefeller Foundation. Additional funds have also been supplied by the Graduate College of the State University of Iowa.

1. Halle: Ueber Störungen der Athmung bei Stotterern, Monatschr. f. Sprachheilkunde **10**:225, 1900.

2. Ten Cate, M. J.: Ueber die Untersuchung der Athmungsbewegung bei Sprachfehlern, Monatschr. f. Sprachheilkunde **12**:247 and 321, 1902.

3. Gutzmann, Herman: Sprachheilkunde, Berlin, 1912.

4. Fletcher, J. M.: An Experimental Study of Stuttering, Am. J. Psychol. **25**:201, 1914.

METHOD

Two Boulitte pneumographs, a Boulitte laryngograph, a modified Boulitte breath pressure apparatus and a Marey cardiograph, each connected with a specially made Marey tambour, served for registration. One pneumograph was placed over the chest and the other over the abdomen. The laryngograph was adjusted to exert sufficient pressure against the thyroid cartilage to insure against slipping. The cardiograph was placed in the fourth or fifth interspace over the cardiac impulse beat. In all but two records, time was recorded in seconds by a Jaquet chronograph. In one of these two exceptions, time was recorded by a 10 double vibration tuning fork, and in the other no record of time was made. The signal line was furnished by a magnetic signal marker. The periods of stuttering indicated on the signal line represent times when external evidences of disturbance of speech were noted. A length of kymograph paper sufficient to record four minutes of talking was used in every experiment. Each subject was used for several experiments, and every observation reported occurred on at least two different occasions.

Throughout the recording period, conditions were kept as uniform as possible. During both the speaking and the resting periods, the subject sat with his back to the kymograph. For the speaking records the subject was asked to discuss in his own language something with which he was known to be familiar. This rarely fails to provoke stuttering.

In all records inspiratory movements of both chest and abdomen, elevation of the larynx and inspiration of air through the mouthpiece are indicated by downward movements of the styli (descending curves on the record) while expiratory movements, depression of the larynx, and emission of air through the mouthpiece are denoted by upward movements of the styli (ascending curves on the record).

RESULTS

Six persons with severer forms of stuttering and several normal speakers from the university student body served in this study. One of the stutterers was an elementary school boy, aged 13; one a junior in high school; one a baker, aged 28, and three were undergraduate students in the university.

The records of normal speech show six fairly fixed relations between movements of the abdomen, thorax, larynx and breath stream (fig. 1): (1) a fairly close correspondence between abdominal and thoracic breathing; (2) the relatively greater number of laryngeal than expiratory movements; (3) the seemingly complete independence existing between the movements of the larynx and the movements of the breath-

ing mechanism; (4) the rhythmic nature of breathing, of the vertical movements of the larynx, and of the breath pressure changes; (5) the marked increase in length of expiration as compared to inspiration during speech and as compared to its own period when speech is not taking place (figs. 2 and 3), and (6) the presentation by the abdomen especially of a number of small in and out movements at a rate, more or less regular, of from 5 to 7 a second.

The synchronism between thoracic and abdominal movements during normal speech is evident in all records. Occasionally one finds instances

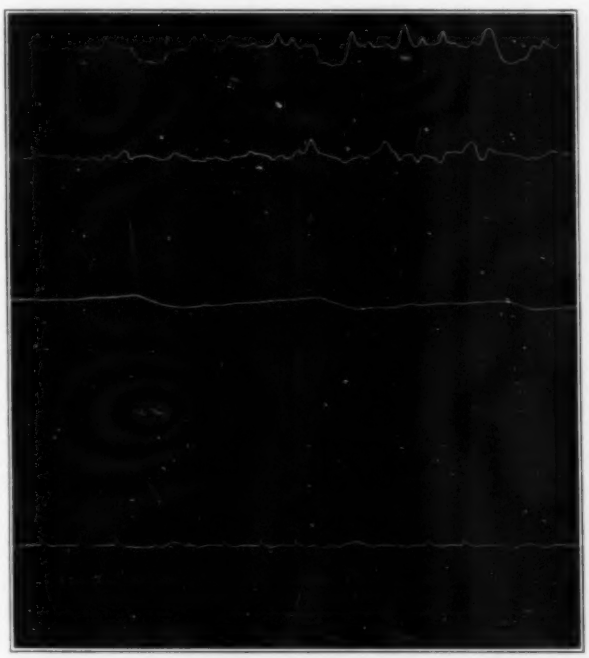

Fig. 1.—Record during normal speech. Reading from the top down, the first curve records breath pressure; the second, vertical movements of the larynx; the third, movements of the thorax; the fourth, movements of the abdomen; the fifth, the heart rate, and the sixth, the time in seconds. The straight line at the bottom is a signal line which is used in following records to indicate periods of stuttering.

of slight anachronisms, in which one of the major breathing units is moving slightly in advance of or behind the other. On the whole, however, it may be said that the thorax and abdomen act as a unit during normal speech.

In regard to the greater number of laryngeal than expiratory movements, actual count shows that the larynx makes on the average six complete up and down movements during one expiration. This organ

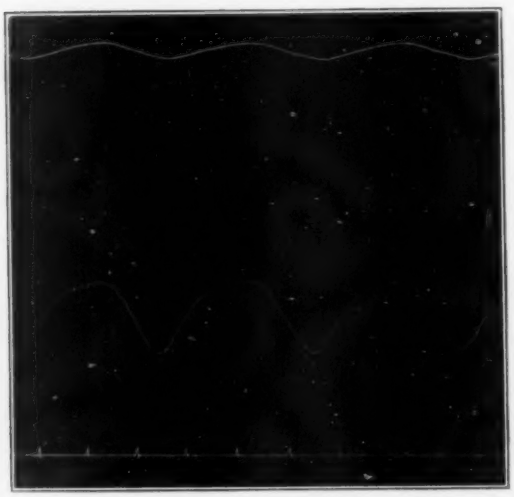

Fig. 2.—Breathing of a normal individual while not speaking. In this figure and in figure 3, reading from the top down, the first curve records movements of the thorax; the second, movements of the abdomen, and the third, time in seconds.

Fig. 3.—Breathing of a normal individual during speech.

is raised and lowered under several conditions during speech. Some of them are articulatory, as in the production of certain sounds which require elevation of the body of the tongue. The larynx is connected by way of the hyoid bone with the tongue and may therefore be raised with it. Also, the larynx may be raised or lowered in the production of nasalized tones that require the soft palate to be depressed. The con-

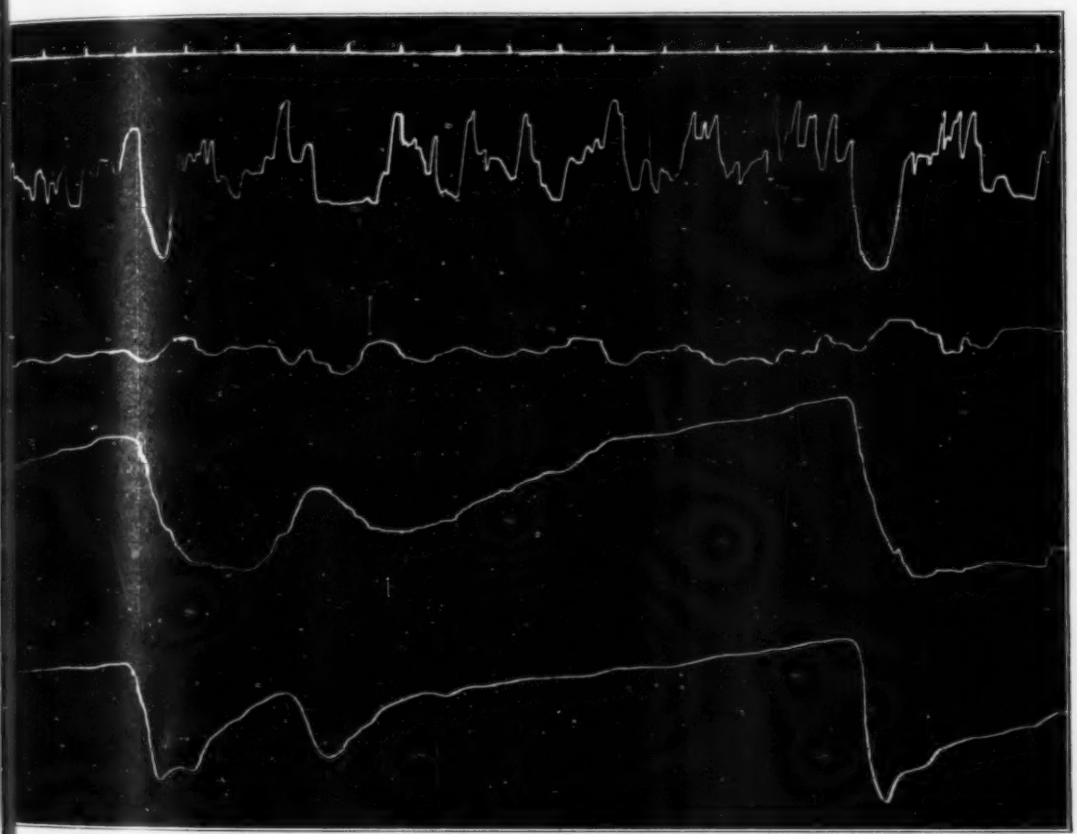

Fig. 4.—Antagonism between movements of the abdomen and those of the thorax during stuttering. From the top down, the first curve records time in seconds; the second, breath pressure; the third, vertical movements of the larynx; the fourth, movements of the thorax, and the fifth, movements of the abdomen. During the period of most marked antagonistic action between the thorax and abdomen, the air column is not moving. The larynx appears to be moving approximately normally.

traction of the palatopharyngeus muscle elevates the larynx at the same time that it depresses the soft palate. Conditions of vocalization, such as the utterance of a high or a low pitched tone, may be responsible for some of the vertical movements of the larynx during speech. The

thyrohyoid muscle elevates the larynx for a high tone, while the sternothyroid muscle helps depress it for a low tone. This method of elevating and depressing the larynx is not common in the speech of a person who has the average amount of pitch variation, but is used often in singing.

In all of these conditions, the vertical movements of the larynx are due entirely to certain of its extrinsic muscles and to certain extrinsic

Fig. 5.—Antagonism between movements of abdomen and thorax during stuttering. Arrangements of curves are the same as for figure 4, except that the time is indicated in tenths of a second. During the period of greatest antagonistic action between abdomen and thorax, the air column is not moving. For the later two thirds of the stuttering period, the muscles controlling vertical movements of the larynx are in tonic spasm.

muscles of the tongue. However, it is possible to elevate this organ by firmly closing the glottis and attempting to force air out of the lungs. This action is brought about by the intrinsic muscles of the larynx and the muscles of breathing. During normal speech, this way of raising

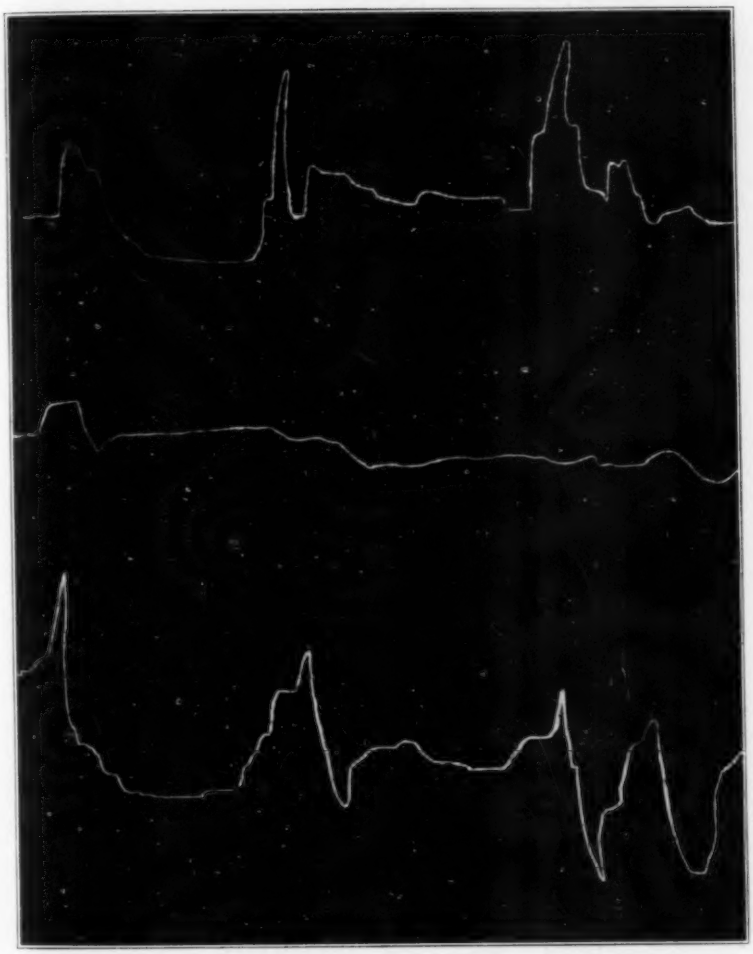

Fig. 6.—Synchronism between movements of larynx and abdomen during stuttering. From the top down, the first curve records vertical movements of the larynx; the second, movements of the thorax, and the third, movements of the abdomen. The subject was stuttering throughout the entire record, which represents about twenty seconds. The thorax is moving asynchronously with respect to the abdomen and the larynx and fails to show a single exhalation.

Fig. 7.—Synchronism between movements of larynx and breathing apparatus during stuttering. In this figure and in figures 8, 9 and 10, from the top down the first curve records breath pressure; the second, vertical movements of the larynx; the third, movements of the thorax; the fourth, movements of the abdomen; the fifth, the heart rate; the sixth, time in seconds, and the seventh, the stuttering period. The contractions of the muscles underlying the cardiograph were synchronous with the movements of the larynx, thorax and abdomen and replaced the apex beat on the record. The larynx is depressed for the slight exhalations and elevated for the slight inhalations.

the larynx is rarely utilized. The glottal stop that occurs in some languages [5] makes use of it to a limited extent.

Thus it would seem that the vertical movements of the larynx are secondary to movements of the tongue and the soft palate and are there-

Fig. 8.—Synchronism between movements of the larynx and breathing apparatus during stuttering. The larynx is depressed for inhalation and elevated for exhalation.

fore to be considered as predominantly a result of articulation; they have been studied here to determine if they play a part in the function of breathing during speech as well as the recognizable parts they play in articulation and phonation.

5. Arabic is said to be the only language that freely employs the glottal stop.

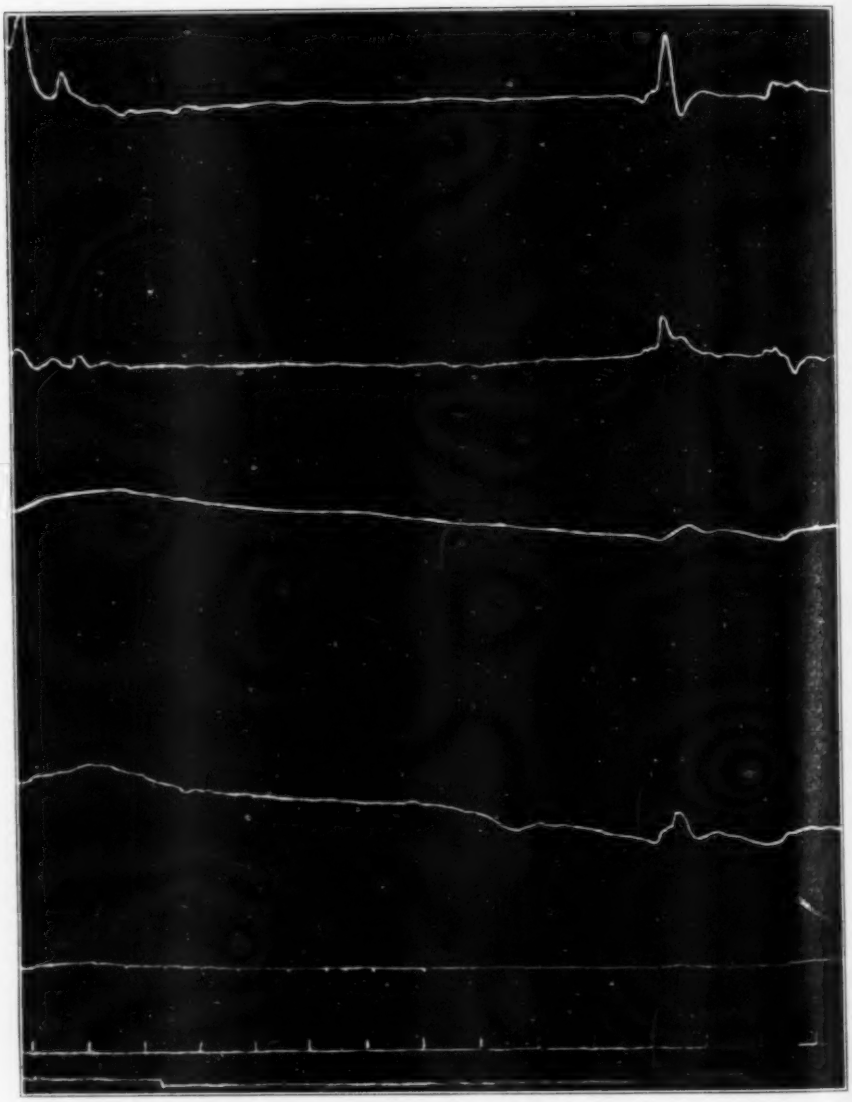

Fig. 9.—Exceedingly long inspiration and tonic spasms of the muscles controlling vertical movements of the larynx during a stutterer's attempt at speech.

There may be an exception to the complete independence between movements of the larynx and those of the breathing mechanism in a tendency in some instances for the larynx to be slightly elevated during

Fig. 10.—Large vertical movements of the larynx during prolonged inspiration in an attempt to speak.

inspiration. On the whole, however, breathing and laryngeal movements appear to be independent of each other.

The rhythmic nature of breathing, of the vertical movements of the larynx and of the changes in breath pressure are characteristic of normal speech. These various movements are not perfectly regular by any

means, but neither long intervals without movements nor periods of excessively fast movements occur.

One of the first things that attracts attention when one is examining records of breathing is the marked increase in the relative length of expiration during speech. There is not much difference in the duration of inspiration and of expiration during quiet periods, but when speech begins the expiratory period becomes comparatively much longer than the inspiratory period.

There can be no doubt in regard to the presence in the abdomen of small in and out movements at a rate varying between 5 and 7 per second. They are more marked in some instances than in others, but they seem to occur with sufficient consistency to be considered characteristic. Whether or not these small movements are present in the thorax is not determined. A few of our records suggest that they may be present.

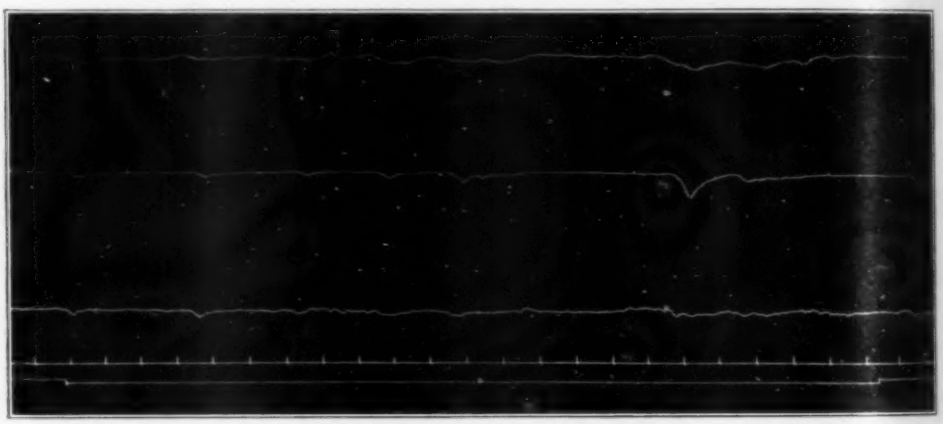

Fig. 11.—Tonic spasms of the breathing musculature and tremors shown on the cardiograph line. (Figure 17 shows an amplification of the oscillations of the cardiograph curve.) In this figure and in figure 12, from the top down the first curve shows movements of the thorax; the second, movements of the abdomen; the third, the cardiograph record; the fourth, time in seconds, and the fifth, the stuttering period. It is impossible to determine the heart rate in this record.

All of these observations indicate that the motor units of the normal peripheral speech apparatus are so integrated as to assure a periodically renewed adequate supply of air evenly provided for the voice producing and articulatory mechanisms.

The stutterer's speech diverges widely from the normal and a wide variety of deviations is obviously possible. Of these, our records afford a considerable number.

One of the most interesting is the diametrical opposition between the action of the thorax and that of the abdomen during stuttering (figs. 4

and 5). That is, the records indicate abdominal inspiration and thoracic expiration at the same time, and vice versa. The two movements, inspiratory and expiratory, carried on simultaneously by the two parts, thorax and abdomen, appear to be of about equal strength and thus tend

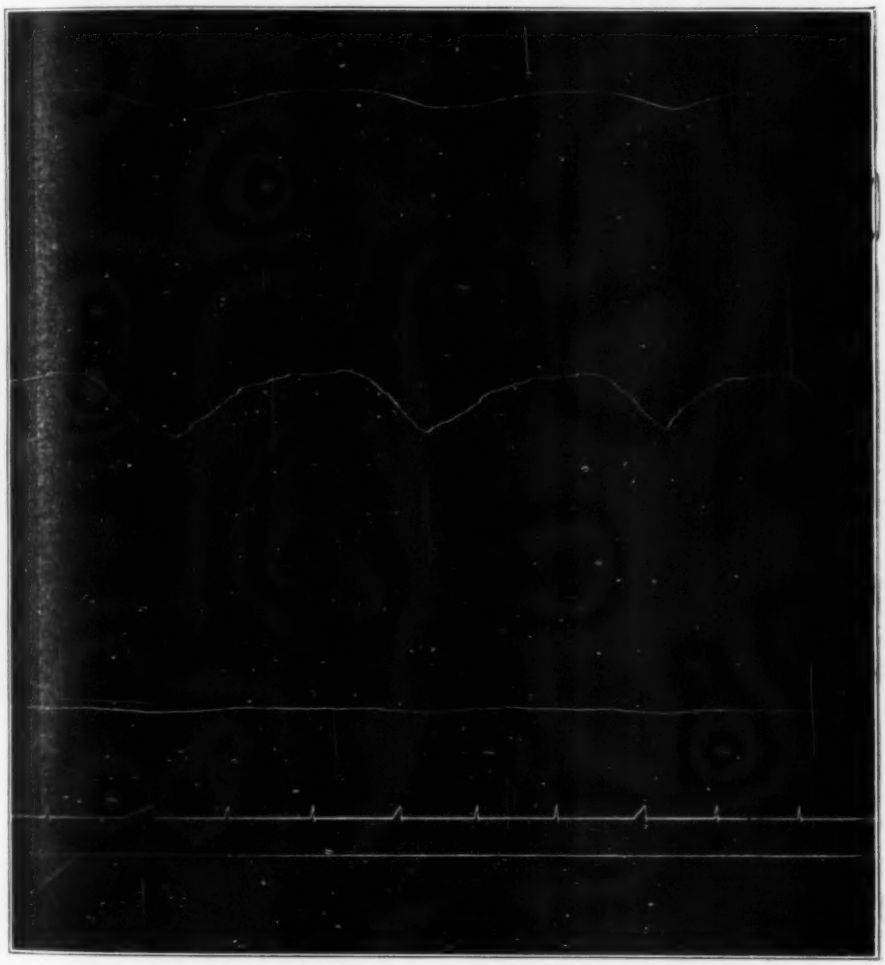

Fig. 12.—Same subject as in figure 11. Breathing without speaking. Here no tremors are recorded by the cardiograph.

to neutralize each other. Hence, during the period when the antagonistic action is the most pronounced the air column is not moving.

Another striking evidence of lack of integration in the speech mechanism during stuttering is the marked synchronism that exists in certain instances between the movements of the larynx and those of the abdomen and thorax. There are several varieties of this type of

synchronous functioning. One is when the larynx and abdomen are displaying a marked similarity of movement while the chest is moving out of time with them (fig. 6). When the abdomen is extended, the larynx is elevated and when the abdomen is withdrawn the larynx is

Figure 13

Figure 14

Fig. 13.—Tonic spasms during stuttering of both groups of breathing muscles and of those controlling vertical movements of the larynx. In this figure and in figures 14 and 15, from the top down, the first curve records breath pressure; the second, vertical movements of the larynx; the third, movements of the thorax; the fourth, movements of the abdomen; the fifth, the heart rate; the sixth, time in seconds, and the seventh, the stuttering period.

Fig. 14.—Tonic spasms during stuttering of both groups of respiratory muscles with the larynx moving normally. Tremors are shown by the cardiograph.

depressed. The movements of the chest seem to bear no relationship whatever to those of the other two units. In some cases in which the synchronism between laryngeal and abdominal movements lasts for several expirations, the thorax fails to indicate a single complete expiratory movement.

A second type of the synchronous functioning of the larynx and breathing apparatus is seen when the abdomen, thorax and larynx make brief simultaneous movements at a rate of about 1.5 a second. In some instances (fig. 7) the larynx is elevated for inspiration and depressed for expiration, while in others (fig. 8) it is depressed for inspiration and elevated for expiration.

Fig. 15.—Tonic spasms of the larynx during stuttering, with approximately normal breathing.

Thus it appears that the larynx during stuttering may move synchronously with the abdomen or thorax or both, instead of showing the independent and faster rate of rise and fall that characterizes normal speech. In this synchronous action, rise of the larynx may coincide with inspiration, and hence its fall with expiration or this relation may be reversed.

A third way in which the stutterer's attempts at speech differ from the speech of normal speakers is marked prolongation of inspiration. Inspiration may continue as long as ten seconds while the larynx is apparently

in tonic spasm (fig. 9). Such periods are frequently terminated by a rather sudden and brief respiratory movement. During some of the periods of abnormally long inspiration, the larynx makes several large vertical movements (fig. 10). In most instances from two to four such movements occur at a slow rate, about one a second.

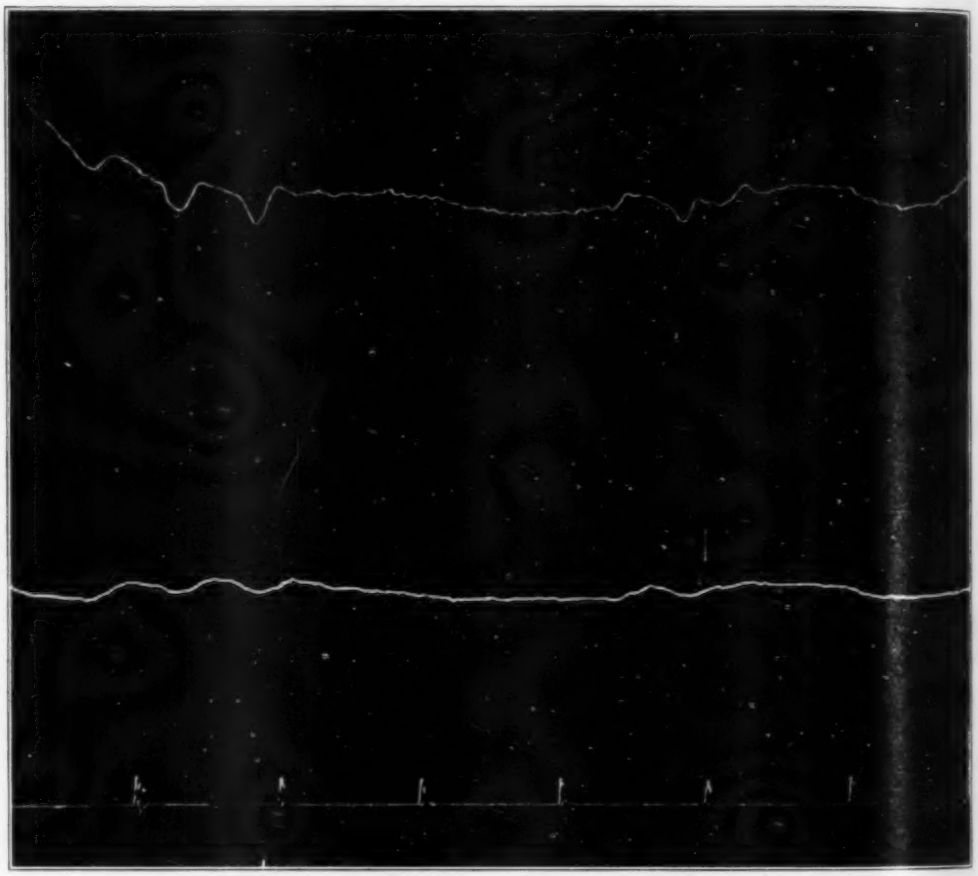

Fig. 16.—Tremors of the abdominal breathing musculature. The upper line records movements of the abdomen; the second, the heart rate, and the third, time in seconds. The stuttering period is indicated by two short vertical lines at the bottom. It is impossible to determine the heart rate in this record. The abdominal breathing record and the cardiograph curve are an enlargement from a record similar to figure 13.

Spasms have long been known to occur in the breathing musculature of the stutterer. They may be of short or of exceedingly long duration and of the clonic or tonic type. One of our cases often presented tonic spasms of the muscles of expiration lasting more than twenty seconds, interrupted with occasional shallow breathing movements (fig. 11).

Fig. 17.—Tremors recorded by the cardiograph during stuttering. Time in seconds. This is an enlargement of the curve presented toward the latter part of the third line of figure 11.

Spasms are not confined to the breathing musculature alone but appear in the muscles of the larynx and in those controlling the organs of articulation. The entire speech mechanism or one or more of its parts may be affected at one time (figs. 13, 14 and 15).

The meaning of tremors in certain movements of parts of the speech mechanism during stuttering is uncertain. Normal speech, as has been pointed out, is characterized by the presentation, on the part of the abdomen especially, of movements at a rate of from 5 to 7 a second. However, during stuttering certain patients may show this rate and in addition a fine tremor of the abdominal musculature with a rate of about 11 per second (fig. 16), which is not shown in the records of normal speech. This same tremor rate is picked up by the cardiograph during stuttering (fig. 17) but not during breathing when speech is not taking place (fig. 12).

SUMMARY

Records of normal speech show an integration of the various units of the breathing mechanism, which exhibits the following characteristics: (1) a fairly close correspondence between thoracic and abdominal breathing; (2) a relatively greater number of laryngeal than of breathing movements; (3) a relatively complete independence between vertical movements of the larynx and movements of breathing; (4) an evident rhythm of breathing, of the vertical movements of the larynx and of the changes in breath pressure; (5) a disproportionate increase in duration of expiration during speech, and (6) the presentation by the abdomen of small in and out movements at a rate varying from 5 to 7 a second.

Our records of stuttering show a dysintegration of certain of the motor speech units which is apparent at various times in the following ways: (1) a complete antagonism between the action of the thorax and that of the abdomen; (2) a marked synchronism between the movements of the larynx and those of the various units of the breathing apparatus; (3) a marked prolongation of inspiration; (4) large vertical movements of the larynx during inspiration; (5) clonic and tonic spasms of the various muscles of speech production and (6) the apparent introduction of a new tremor rate in the abdomen.

UNILATERAL (UNASSOCIATED) INNERVATION OF THE OCULAR MUSCLES *

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The case studied and reported in this paper was one in which normal associated lateral ocular movements were present, but, in addition, the patient had the power of maintaining fixation of either eye on an object directly in front of him while he slowly rotated the other eye widely outward and brought it back on command.

This condition has been described in various ways, and the entire literature is not extensive. With the exception of the brief reference to Gould's case, all the literature on the subject is in the German language. I have not found a single paper written in English or in French. The phenomenon has been regarded as exceedingly rare by all authors except Schwarz,[1] Levi,[2] Lechner,[3] and possibly Bielschowsky,[4] but what they describe is different in some respects from the movement that has interested me, although it is important. They describe movement merely of one eye alone from the median line inward, while the other eye remains in the primary position. This seems to be a condition that can be acquired only by some of those persons who have the power of converging the eyes without fixing on an object, and it requires special training. I have examined two persons who had this power of convergence without fixation, both ophthalmologists, but they did not have the power of moving one eye alone inward while the other remained fixed. In this form of movement, the converging eye merely returns to or near the primary position, and does not deviate widely outward, as it does in the other cases to which I refer.

The cases I have found reported in the literature, omitting the reports already mentioned of the movement described by Schwarz, Levi and Lechner in themselves, are eight in number (Königshöfer,[5] one case; Peters,[6] three cases; Gould,[7] one case; Lechner, 2 cases, and Kolen,[8]

* Read before the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

1. Schwarz: Centralbl. f. prakt. Augenheilk., vol. 21, p. 107.
2. Levi: Klin. Monatsbl. f. Augenheilk. **46**:167, 1908.
3. Lechner: Von Graefe's Arch. 1887, vol. 44, Klin. Monatsbl. f. Augenheilk. **53**:168, 1914.
4. Bielschowsky: Pflüger's Arch. **136**:658, 1910.
5. Königshöfer: Bericht über die 25 Versammlung d. Ophth. Gesellschaft. Heidelberg, 1896, p. 313.
6. Peters: München. med. Wchnschr., 1905, p. 2205. Wesenberg, Dissertation, Rostock, 1906; Klin. Monatsbl. f. Augenheilk. 1907, vol. 45.
7. Gould: Section on Ophthalmology, College of Physicians of Philadelphia, Meeting of April 16, 1907; Ophthalmic Rec. **16**:400, 1907.
8. Kolen: Arch. f. Augenheilk. **97**:341, 1926.

one case). I shall not discuss voluntary unilateral vertical movements, as I have never had the opportunity to study a case of this type. It is considered carefully by Bielschowsky.

It is interesting to note the titles employed (translated) by the different authors describing this phenomenon in the order of publication of their papers: Königshöfer employed the title "A case of voluntary movement of the left eye in horizontal direction with primary position of the right eye." Schwarz used as title "Concerning voluntary unilateral ocular movements." Peters used "Concerning voluntary squinting [Schielen] of one eye with primary position of the other eye." Levi used the same title as that employed by Peters. Lechner used "Abnormal voluntary ocular movements." Bielschowsky used "Unilateral (unassociated) innervation of the ocular muscles." Kolen used "A contribution to the question of unilateral voluntary ocular movements."

It is interesting to note that Königshöfer employed a title which in no way committed him to an explanation, and he attempted no explanation. Schwarz, Bielschowsky and Kolen all employed terms indicating unilateral movement or unilateral innervation, and all three attempted to prove that the movement was not unilateral, but that it conformed to Hering's law concerning the equal innervation of both eyes. Even though one may grasp the force of what they say in this interpretation, at least for voluntary inward movement, it is difficult to understand how the innervation of the two eyes is equal in the outward movement of one eye alone.

With the exception of the patient studied by Kolen, probably every other one had strabismus. In Kolen's patient, the statement is definitely made that the eyes were normal. There can therefore be no question in this case as to the occurrence of voluntary unilateral movement without ocular defects of position of the eyeballs and of associated binocular movements.

Every explanation attempted, with the exception of Kolen's, has depended on the mechanism of ocular movements and, although the condition was first recognized in 1896 (Königshöfer), Kolen, writing in 1926, stated that doubt exists concerning the value of the explanations as yet offered for voluntary unilateral movement, and added that the phenomenon is far from being satisfactorily explained.

It is desirable to consider briefly the cases reported in the literature.

Königshöfer's case was one in which voluntary movement of the left eye in a horizontal direction inward, directly forward or outward was done on command with the left eye, without associated movement of the right eye, and with fixation of the right eye in the primary position (median position).

A man, aged 24, had always had divergence of the left eye and poor sight in this eye (strabismus divergens). Königshöfer said that as his case was the only one in the literature he would not attempt an explanation. He presented his case as a curiosity. Schwarz said of the case that the ability of the patient to bring the eye in absolute divergence doubtless depended on the fact that the physiologic position of rest was divergence. It seems improbable that the divergence is to be explained in this way, for while it was helped in the manner described, Kolen's case proves definitely that the movement into divergence was not dependent on any form of strabismus, as his patient had normal eyes.

The case reported by Peters, in 1905, and later by Wesenberg from the service of Peters, was of a patient with poor vision in the left eye and good vision in the right eye. It was not known how long this movement had existed. The left eye was in divergence. Convergence was preserved. When the man was told to look inward with the left eye, this eye went from divergence far inward, so that the nasal edge of the cornea touched the inner canthus, but the right eye remained in the primary position. The patieht could do this in far or near vision. The pupil contracted in inward movement.

Two cases were reported by Peters in 1907. In both, divergent strabismus was present in the left eye. In both, the left eye could be moved inward and then outward at will with fixation of the right eye in the primary position. He explains the condition as an exception to the law of equal innervation of Hering and as the result of the passage of the accommodation impulse only to one rectus internus, which was stimulated in this way to unusual effort and which accomplished what both internal recti usually accomplish together. This explanation has not been accepted by others who have written on the subject; indeed it has been vigorously combated by Schwarz, Levi, Lechner and others. In two cases reported by Peters, voluntary inward movement was associated with bilateral and equal accommodation and contraction of the pupil.

Schwarz had no case. He says that after practice and without definite fixation he was able to perform unilateral convergence and relative movements of divergence.

Levi tries to prove that in the cases described by Peters the innervation was not strictly unilateral, although Peters says of his first case (1905): "The right eyeball stays immobile in the median line." In his two other cases, in association with inward movement of the left eye to a position of moderate convergence, the right eye showed narrow movements to the left and right. This, Levi concludes, indicates first adduction and then abduction in the right eye and proves that *equal* [the italics are mine] double innervation existed, and establishes the correctness of Hering's law. However, it is difficult for me to understand in what way the innervation of the two eyes was equal.

Levi was able to move his eyes horizontally separately; he had strabismus divergens latens of each eye, so that he could produce or overcome the strabismus at will. When he fixed one eye, he could move the other inward to convergence. He believed that the less developed the binocular muscular apparatus is from birth, the more easily the unilateral voluntary divergence and convergence can be accomplished. It was possible, he thought, that the reason Lechner and Schwarz were able to perform unilateral muscular movements inward with comparatively little effort was that binocular vision was imperfect with them. Lechner stated that he had a moderate strabismus divergens for near objects, but Schwarz made no such statement concerning himself.

Lechner disputed the great rarity of unilateral movements, although Königshöfer had considered his case unique. Lechner asserted that he knew several persons with normal eyes who had this movement of one eye, but he seems to be speaking only of movement inward from the normal median position of the eyes. He himself learned after some practice to accomplish this movement. Without covering the eye he wished to move and without holding the finger for fixation before the other eye, he was able to move one eye inward. He regarded this as more difficult to accomplish with a normal eye than with an amblyopic eye. When he paralyzed accommodation of the fixing eye with homatropine, as Königshöfer had done in his patient, Lechner was able to move the other eye with less effort. He found a case in which accommodation was absent during unilateral movement (he is always speaking of this inward movement), and he considered that this supported Hering's law. All these details he published in 1897. Absence of accommodation would show that unilateral movement inward is not dependent on accommodation but that it can be acquired by practice. Peters rejected the opinion that this inward movement may depend on training, but believed that, instead of normal convergence, an incoordinated innervation had existed from early youth.

Lechner, in his paper, published in 1914, reports a second case in which divergence of the eyes and then fixation on a point were possible without the least accommodation, as in his first patient (Vermet). This he regards as a great curiosity. His second patient, a physician (de Flines), had strabismus from youth which he could overcome. His abduction and adduction of both eyes occurred without the least accommodation, although the pupils became smaller in convergence. In both of Lechner's cases, it was possible without covering one eye to allow one eye to diverge and to bring it back. The patient could do this as slowly or as quickly as he wished, but the photographs in the second case show moderate outward deviation of either eye, and there is no mention of isolated inward movement beyond the midline. Lechner thought that Vermet's case was more nearly a strabismus latens, because the eye

deviated when it was covered and could not be prevented from doing so, as occurs in strabismus latens. This was not true of de Flines, who behind the covering hand could allow the eye to diverge and could bring it back, always without accommodation. In Vermet, the voluntary movement in large part depended on the will; this was not so with de Flines, who had the movement in each eye, while Vermet had it only in one eye at first and learned to move the other eye by practice.

Lechner does not make it clear what he considered the cause of the outward movement of the eye. The isolated movement of the left eye was slight in the photograph of de Flines; the isolated movement of the right eye was greater but was apparently less than in my case. He speaks of the outward movement as voluntary squinting (schielend) in the legends of the photographs of de Flines.

Bielschowsky did not report any case of horizontal unilateral movement of one eye, although he observed vertical unilateral movement. He says that the statement from Ewald Hering concerning the equal innervation of both eyes has been proved correct in so many and in such different disturbances of movement that in every case of unilateral or unequal ocular movements one must ask whether these movements are not produced by the combination of different but equal innervation of both eyes. Many isolated movements are described incorrectly as unusual phenomena contradicting this law of association of binocular innervation, in spite of the fact that the signs are easily demonstrable by which the bilateral innervation is recognizable as the foundation of the unilateral movement. He who can contract and relax his muscles of convergence at will, without the use of an object of fixation, after some practice will be able to bring the right or the left eye from the middle position separately inward and then to permit it to return to the middle position. He needs only, when he fixes an object in front of him and not too near, to innervate his eyes in convergence, and when diplopia appears to concentrate his power of fixation of one eye on the object. If this is done, for example with the left eye, this eye will remain stationary and only the right eye will wander inward. If the convergence innervation with the same object of fixation is given up, the right eye will return to its former position.

The occurrence of unilateral ocular movements is in no way dependent on anomalies of motion or of position of the eyes, and those which occur under such circumstances are distinguishable chiefly from those which occur under normal conditions only in degree and direction of movement. When a person can move one eye at will outward or upward, it does not follow that the movement is accomplished by isolated innervation of this eye. A condition favoring the occurrence of such an unusual movement is a corresponding anomaly of the so-called anatomic position of rest. If the position of rest is one of divergence,

the complete relaxation of convergence innervation of one eye, while the other remains fixed, causes the first not only to pass from the previous position of adduction to the middle position, but also to be abducted beyond this. It is easily recognizable that a person with a divergent eye, who wishes to bring this eye parallel to the other eye, uses binocular innervation for focusing at the near point, by the fact that the unilateral movement is associated with bilateral equal increase in refraction and narrowing of the pupil.

Bielschowsky thus explains the movement of one eye inward or outward as the result of contraction or relaxation in convergence innervation, but while there is bilateral innervation, as shown by the change in the pupil and in accommodation, the relaxation of the internal rectus does not seem to be sufficient to explain the slow extensive movement of the eye outward, especially when convergence is almost nil, as in my case. The deviation of either eye outward in my case, as I have determined recently in two cases, is considerably greater than occurs in complete oculomotor paralysis, in which the contractile power of the internal rectus is abolished. There is bilateral innervation, as shown by the bilateral dilatation of the pupil in unilateral outward movement of the eyeball and in the bilateral contraction of the pupil in unilateral inward movement, but it seems to me that the outward movement of one eye is not merely a passive act by relaxation of the internal rectus, but is an innervation movement through contraction, in part at least, of the external rectus. This view is supported by Dr. Holloway, as he states that the outward rotation of each eye in this case is dependent on a definite external rectus action. The rotation is so extreme that it could not be dependent merely on a relaxation. It is well beyond the angle of deviation noted under pronounced anesthesia when the divergence of the eyes always occurs. Kolen has written the most recent (1926) paper on unilateral lateral movements, and with full knowledge of the literature on the subject asserts that they are exceedingly rare. He says that almost every case that has been reported has led to a discussion of Hering's law. Each author tries to explain the mechanism of the monocular movement.

Kolen's patient had much to do with stereoscopic photographs, and without any instrument he was able to fuse both stereoscopic pictures into one. He did not know when he began to have isolated movements of the eyes, but thought it was in childhood. The ocular movements were not limited in any direction. While one eye was in the primary position, he could move at will the right eye inward or the left eye inward or outward. If one eye was covered by the hand and the other fixed on a near object in the median line, the covered eye preserved the primary position and made no inward movement after the hand was removed, so that diplopia occurred, which he could overcome voluntarily

by a slow or rapid movement of convergence. At will he could make the proper inward movement with the covered eye, so that when the hand was removed both eyes were on the point of fixation. The eyes of the patient were entirely normal. Cold water in the ear caused nystagmus of both eyes. Kolen thought that if dissociation of bilateral innervation existed it would be demonstrated by this test. The reaction was entirely normal, and bilateral nystagmus was produced from either ear. He considers anomalous position of the eye in the orbit, latent strabismus and weakening of the fusion effort as important for unilateral movement, and yet as his patient had normal eyes it would seem that these conditions were not indispensable. He is willing to believe that unilateral innervation later may possibly be accepted in many cases of voluntary unilateral ocular movement but thinks that it was not to be accepted in his patient. All these isolated movements require a considerable increase of attention. In his opinion, no explanation as yet offered is satisfactory. The association of ocular movements is a conditioned or combined reflex. All these reflexes can easily be inhibited or intensified. The ocular movements usually are produced by irritation of the light perceiving apparatus of the retina. The significance of concentrated attention in causing conditioned reflexes is universally known, and this explains the results obtained by Lechner and Schwarz on themselves. Kolen thinks that unilateral ocular movement is not to be explained by anomalous innervation of the ocular muscles or by anomaly of the position of rest, but by the development of a new conditioned reflex. An anomalous position of rest or a muscular insufficiency renders this reflex more easily acquired. The statements of Lechner and others that these movements may be acquired by training can be harmonized with the theory mentioned. This view, he says, needs further experimental investigation and is at present nothing more than an unproved theory.

My attention was first directed to unilateral ocular movements in 1907, and ever since that date I have been on the watch for a case of this character, but have never found one until recently. At a meeting of the Section of Ophthalmology of the College of Physicians, April 16, 1907, I read by invitation a paper on paralysis of upward associated ocular movements. In the report of this meeting, it is stated: "Dr. Gould referred to a patient who was able to turn either eye at will outward while fixing with the fellow, and was able to read test cards placed 90 degrees from each other without altering the position of the diverging eyes. There was limitation also of the upward and downward movements and a slight deviation outward of one eye in ordinary fixation, for which operation had been proposed. He asked Dr. Spiller if he knew of an explanation for such a condition. Dr. Spiller answered Dr. Gould: 'The only interpretation, if the isolated movement of the eyeball was

active and not passive, would seem to be that the individual was extraordinarily developed, and must have a cortical center which controlled the movement of one eye outward independently of the other eye. Such a condition had never been demonstrated.' "

REPORT OF CASE

My case is as follows:

H. U., a colored youth, aged 20, consulted me, Feb. 28, 1927, complaining of tremor of the tongue and of the right hand. He had noticed these symptoms for two years and because of them had been obliged to discontinue attendance at high school, where he was a senior. At this time he had what was called a "nervous breakdown." The symptoms have gradually become worse. His mother noticed,

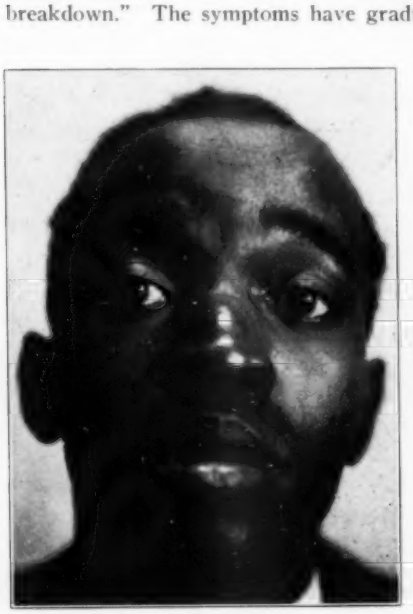

Figure 1

Figure 2

Fig. 1.—Appearance of patient when looking directly forward with the visual axes about parallel.

Fig. 2.—Normal associated movements of the eyeballs to the right.

in September, 1926, that his speech was becoming affected, and was what she called "thick." The tremor of the right hand was more pronounced when he wrote or made special movements. In 1920, he could not sleep at night for six months, but would fall asleep in the daytime. He never had fever, never had any acute illness that he can remember and never had diplopia. He is an only child. Weakness of the limbs was not present. The tendon reflexes were normal. The tongue was in constant coarse twisting movement, and fine tremor could be seen in the lips and the right side of the face.

The Wassermann reaction of the blood in the first test, made February 8, was positive with three antigens; the Kolmer test was negative and the Kahn test weakly positive. Other tests made later gave negative Wassermann and Kahn

reactions. The epitrochlear and postcervical glands were palpable. Though the ocular condition was the interesting feature, he had not come to the dispensary because of it.

When he looked at any object, both eyes were in the median position, but if he was not fixing an object, the left eyeball had a tendency to rotate outward a little, but did not always do so. If he saw that some one was watching him and he looked at the observer, the left eyeball at once came into the median position. When told to turn the left eyeball out, he moved it slowly to the outer canthus and the right eyeball stayed in the median position. When told to move the right eyeball out, he moved it slowly, equally as far to the outer canthus as he had moved the left eyeball, and the left eyeball stayed in the median position. Isolated movement was easier for him in the left external rectus muscle than in the right. When he looked upward, the eyeballs diverged a little.

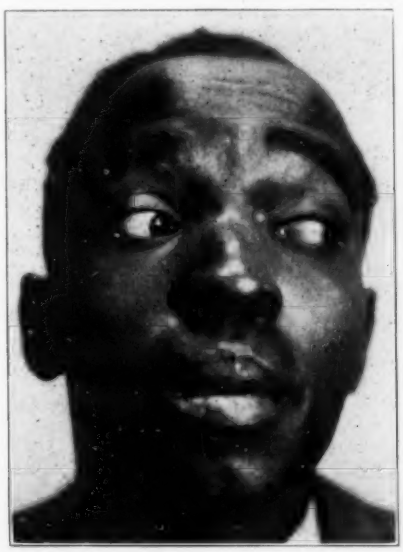

Fig. 3.—Normal associated movements to the left.

Downward movement seemed good. It was striking how the left eyeball tended to deviate to the left when he was not looking at an object and the promptness with which it returned to the median position when he fixed an object. He could bring the left eyeball from the extreme left slowly, but he brought the right eyeball from the extreme right quickly, not slowly as he brought the left eyeball from the extreme left. This might possibly have been the result of a little weakness of the right internal rectus muscle. He probably had had this separate voluntary movement of the eyeballs all his life, as the mother said that the pupil of the left eye "had been farther to the side" all his life, but the unilateral voluntary movements were never recognized by any one, even by the patient, until I asked him to try to turn each eye outward separately. The associated movements to the right or to the left were normal. Under cover, the eye did not always deviate outward, but frequently did, and when it had deviated it could be brought back to the midline at will and could be moved outward again

under cover. When it had deviated under cover as far as it usually did, it was still less deviated, to a considerable degree, than when he voluntarily moved the eye from the median position outward. When the eye had deviated outward under cover, he was unable on request to move it further outward to the extent to which he moved it voluntarily from the median position. After repeated tests of the unilateral movement, a weakness in the extent of the excursion outward had been noticed; this was evidence of innervation of the external rectus muscle and not merely of relaxation of the internal rectus muscle, for fatigue is not a property of relaxation.

Although he had been given glasses by some one I do not know, he informed me that no one had ever remarked on the peculiar ocular movements, and he was not aware that he had them until I called his attention to them by testing his power of unilateral movement.

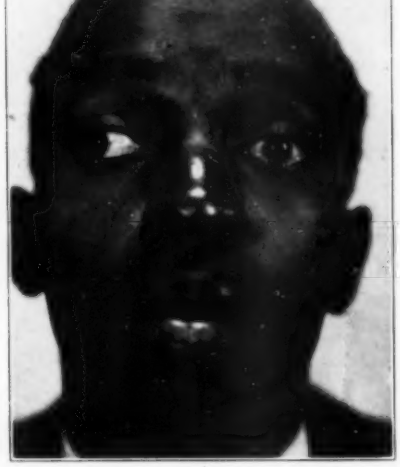

Figure 4

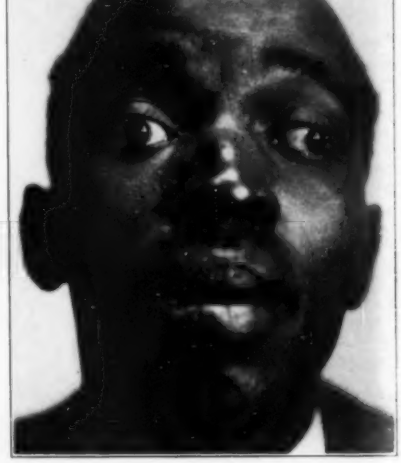

Figure 5

Fig. 4.—Voluntary movement of the right eye to the extreme right while the left eye remains in the median position.

Fig. 5.—Voluntary movement of the left eye to the extreme left while the right eye remains in the median position.

COMMENT

The man appeared to have alternating strabismus divergens, which was remarkably under the control of voluntary movement. I am inclined to believe that the outward deviation was always voluntary and purposive, although it may have become a habit, and the movement at times was unconsciously performed. He had monocular vision, shown by holding a pencil in front of a page which he was reading, when a part of the lines obscured by the pencil was not recognized, as would not be the case if he had had binocular vision. His power of bringing

the eye deviating under cover back to the primary position while it was still under cover is suggestive of voluntary innervation and is not what usually occurs when the eye deviates in strabismus when the deviating eye is covered. I believe that the outward movement of either eye was partly the result of relaxation of the contraction of the internal rectus, which in this case must have been comparatively slight, as convergence was almost absent, but relaxation of the internal rectus was not enough to explain the slow movement of the eye outward until the outer edge of the cornea touched the external canthus, i.e., slow movement if the patient was told to move the eye slowly, but quick movement under the control of his will if he was told to move the eye outward quickly. Kolen's case also is evidence of innervation of the left external rectus, because there was no strabismus in his case, and the outward movement was not the outward "drifting" of a squinting eye returning to its position of rest.

The history of inability to sleep at night during 1920 for a period of six months, when epidemic encephalitis was prevalent, and the tendency at that time to fall asleep in the daytime; constant rolling or twisting movements of the entire tongue of coarse character, tremor of the muscles of the lips and chin, tremor of the right hand, all symptoms which had gradually increased during the past two years, with more recent development of "thickness" of speech, all make the diagnosis of a former attack of epidemic encephalitis with progression of symptoms probable, and explain the almost complete absence of convergence, which is frequently the result of epidemic encephalitis. This case seems to be the only one in the literature in which unilateral ocular movement existed with almost complete absence of convergence and no binocular vision.

Dr. T. B. Holloway examined the eyes. His report is as follows:

Upon rotation to the left, there seems to be slight weakness of the right internal rectus. All other rotations are full. Upon downward movement, rotation and fixing there is marked tendency for the left eye to deviate outward. Upon upward rotation and fixing, one eye or the other is usually deviated outward. There is no true nystagmus. Under cover each eye is deviated outward at least 35 degrees. The orbicularis is intact. The palpebral fissures measure 8.5 mm. The pupils are equal, 2.5 mm. in moderately bright light. The pupils react to direct, indirect light, accommodation and on attempt at convergence. Convergence is extremely poor, in fact practically nil. When fixing with O. D. upon request, the left eye can be permitted to deviate outward so that the external limbus of the cornea comes within 1.5 mm. of the external canthus. Upon request, when fixing with O. S. the right eye can be allowed to deviate outward to the same extent. [Dr. Holloway states here that the eye would deviate outward to within 1.5 mm. of the external canthus. At times, when the patient was not tired I have observed the eye to deviate fully to the external canthus.]

Right eye: Media clear, the disk is nearly round, not suspicious of any pathological change. No changes in the choroid or retina.

Left eye: Similar.

The patient is definitely monocular. It was impossible to work out a satisfactory diplopia field.

Right eye: —.25 ax. 90°=6/6.

Left eye: +.12 sph. = 6/6?

I am quite in accord with you in regard to the independent action of the externa. I believe at first there is a unilateral relaxation followed by a unilateral contraction of the externa. I certainly would incline to congenital origin.

When the patient fixes for distance and the visual lines are as near parallel as they can be maintained, a certain amount of sphincter tone is evident. When one eye is permitted to drift outward there occurs a slight dilatation of the pupils. When the eye returns to fix a distant point, a contraction of the pupils occurs. With the pupils slightly dilated and with one eye deviated outward, if the patient is asked to fix a test object at the reading distance, a greater contraction of the pupils occurs than when the eye is brought to parallelism to fix a distant object.

His accommodation is intact in each eye, and he accommodates quite well with either eye fixing. When either the right eye or the left is allowed to deviate outwards, he cannot accommodate with the deviated eye, but invariably will bring the eye straight forward or in the usual reading position to perform this function. In other words, we could not elicit accommodation with either eye when it was deviated. It seems to me that the explanation of the above phenomena is as follows:

This patient has a marked tendency to a deviation outwards of the visual axes and when attempts are made to bring the eyes as near as possible to parallelism, there is a certain amount of sphincter tone maintained. When his eye is allowed to deviate the associated action between the internus and accommodation and convergence is relaxed, and a slight dilatation of the pupil occurs. In turn, when the eye is brought into parallelism again the interni act, that of the deviating eye the more, at the same time the other one is called into play to a certain extent, and with it an impulse goes to the sphincter, causing slight contraction of the pupil. If this is carried on to a further degree where there is an actual attempt at convergence, we then have brought into play the usual association between that of convergence and accommodation, and we obtain a greater contraction of the pupil.

When we attempted to test his accommodation of one eye when it was deviated, as above stated, we were unsuccessful, and in order to read fine print the deviated eye would invariably be brought to the midline.

Dr. Holloway did not believe that there was a dissociation between accommodation and convergence, but later, when the man was examined by Dr. Adler with the retinoscope, as described below, this dissociation was demonstrated.

Dr. Francis H. Adler also gave much time to the examination of the eyes. His report is as follows:

This boy has the ability to fix an object at, let us say, 6 meters distance with both eyes but at will either eye can be turned out while fixation is maintained with the fellow eye. He has not the ability to turn either eye in from the midline during such fixation. He can, however, at will bring back the deviated eye to its original position of fixation with its fellow.

The field of rotation of each eye is full in all directions save that of adduction and when the two eyes are tested together the rotations are full in all

directions save for convergence. With a maximal effort, both eyes show some convergence but it is certainly far less than normal.

When he allows one eye to deviate outward while fixing an object at 6 meters distance, both pupils dilate and to an equal amount, as far as I could observe. When the deviating eye is brought into its primary position both pupils constrict and to an equal amount, so far as I can observe. The same thing is true as regards the pupillary changes when the patient is fixing an object at 33 centimeters, i. e., the pupils dilate when one eye is rotated outward and constrict when the eye is brought back into position. The patient has no binocular vision and when one eye is turned outward the visual image pertaining to this eye is mentally suppressed.

While fixing with one eye and deviating the fellow eye outward, the patient can continue to read print held at the normal near point of the fixing eye. At the same time it can be shown that the deviating eye accommodates like the fixing eye. I have tested this in the following manner:

The pupils of both eyes were dilated with euphthalmine, three drops, together with two drops of 4 per cent cocaine solution. The patient was instructed to look straight in front of him and while so doing to turn one eye outward. A retinoscopic examination was then made on this deviating eye. With the eyes held in the same position print was placed at the normal near point in front of the fixing eye, and the patient was instructed to read. The retinoscopic examination of the deviating eye now showed very clearly an increase in refraction of about 2 D. The exact amount, however, could not be determined. This test was done on each eye with the same result. It follows, therefore, that even when convergence is in abeyance or is actively overcome by a strong movement of abduction on the part of one eye, accommodation, i. e., constriction of the ciliary muscle, occurs in both eyes to approximately an equal amount during the normal act of reading. What does this indicate? To my mind it shows that this man has developed a marked power of dissociation between the act of convergence and the act of accommodation. He can constrict his ciliary muscle while his convergence is held in abeyance or, as I have stated above, is prevented by a voluntary movement of abduction. Although this dissociation is not seen under normal conditions it is easy to demonstrate physiologically if one places prisms in front of each eye in such a way as to obviate the necessity of turning the eyes in to see print held close to the eyes. With such prisms in place, print can be clearly read at the normal near point even though the visual axes are parallel. In other words, the individual is contracting his ciliary muscle but is not contracting his internal recti muscles. I do not know of anybody who can do this without the aid of prisms, but the opposite of this condition, I know, is normal to some people and in fact is often practiced voluntarily by children. By this I mean the ability to converge the eyes strongly without any accommodative effort. I myself can at will converge my eyes without looking at any near object, and when I do this I develop immediately an homonymous diplopia.

To summarize then, this patient has a voluntary dissociation of the external ocular movements in one direction of gaze. As would normally be expected, the relaxation of convergence power (tone of internal recti) leads to a dilatation of the pupils and a return of convergence power to a pupil constriction. There is an unusual amount of dissociation between convergence and accommodation. At first I was inclined to explain the whole picture on the following basis—that the patient had a very high exophoria which, let us say, was practically a true divergent squint but which could be still overcome by an excessive effort on the part of the internal recti muscles. The act of deviation of each eye separately could then be explained on the basis of an inhibition of the tone of one or other

internal rectus, in which case it would be appropriate to use the term "drifting outward" for the deviating eye. This in itself would be unique, for as far as I know the usual teaching regarding ocular muscle function is that the impulse is always bilateral in the case of associated muscles. It is true that by the law of reciprocal innervation turning the eyes to the right, let us say, implies a constriction of the left internal rectus together with an inhibition of tone of the right internal rectus, but this is not a case in point. Here we would have (if our assumption of tone were the whole story) an inhibition of tone on the part of one internal rectus with no change in tone of the opposite internal rectus.

On the basis of the tests which you proposed and carried out, I am inclined to change my whole opinion. I can assert that when one eye is covered while he is fixating an object 6 meters distant the deviation of the covered eye is not as great as when both eyes are uncovered and starting from the midline he voluntarily turns that eye outward as far as it will go. This would certainly indicate to my mind very strong proof that in the act of deviating the eye from the midline we had to do with not only a cessation of internal rectus tone but also an active constriction of the external rectus muscle. As further evidence of this it was quite apparent that repeated attempts to fix with one eye and turn the other eye out resulted in a gradual diminution in the extent to which the eye could be deviated. I infer from this that the external rectus muscle gradually becomes fatigued from the voluntary effort.

It would, of course, be valuable to have further proof that an active constriction of the external rectus muscle occurred, but the methods by which this could be obtained would probably be quite involved and require much time and experimentation. As an instance, I would think immediately of attempting to record action currents from the external rectus muscle by the string galvanometer just as one can record action currents in constricting voluntary muscles elsewhere in the body. This man shows, therefore, the ability to control the movement outward of each eye independent of its fellow by means of a relaxation of the internal rectus muscle and an active constriction of the external rectus. I am extremely puzzled to know why he has so little convergence. [This was explained to Dr. Adler later as possibly the result of epidemic encephalitis.] Does this suggest anything to you, because it certainly is significant?

Regarding the question you asked as to whether an individual who had, let us say, an ordinary exophoria and whose eye deviated under cover, could voluntarily bring the deviating eye back to position while still under cover, my answer would be no. I admit complete ignorance, never having thought of trying this, but my reasons a priori are these:

The knowledge of the position of our eyes in our orbits is probably mainly obtained from other factors than the proprioceptive impulses coming from our eye muscles. When an eye deviates under cover, therefore, the individual is not aware to begin with that the eye has undergone any change of position. He would not, therefore, be able to tell which way to turn the eye should you suggest to him that he return the eye to its normal position. It goes without saying, however, that in making this test, if you should carry it out, extreme care would have to be observed not to tell the individual to turn the eye in because as I noted, some people have the ability of converging the eye at will without actually fixating a near object.

Until the present, all the cases of voluntary unilateral ocular movement have been reported by ophthalmologists, and all have sought an explanation in the mechanism of ocular movement. Kolen alone has

expressed dissatisfaction with the theories offered and has stated that they do not explain the condition properly; he even seems to revert to the view of Peters, so much disputed in the past, viz., that unilateral movement possibly may be strictly unilateral and contrary to Hering's law, although he does not accept this explanation for his own case. Kolen departs from the mechanical explanation in suggesting the theory of the conditioned reflexes, but acknowledges that it is an unproved theory.

No one seems to have considered voluntary outward movement as a reversion phylogenetically to an earlier type of development or as the result of innervation from a cortical center having control of such a movement, as I suggested in 1907.

E. Treacher Collins,[9] in his Bowman lecture, says:

> The most laterally placed eyes are met with among the *Rodentia*, the *Marsupialia* and the *Ungulata*. The greatest divergence is in the hare, whose optical axis in each eye measures 85 degrees of divergence from the middle line. It seems probable that they, and some of the other rodents, have complete panoramic vision, i. e., are able to see in their circumference at one and the same time.
>
> Rodents have no conjugate movements of the eyes, and no consensual response of the pupils to light, only a direct reaction.
>
> Conjugate movements of the eyes in the vertical plane are innate in the human infant, but in the horizontal plane are only gradually acquired during the first six months of life.
>
> From the early development of conjugate movements in the vertical plane, we may infer that man's mammalian forbears were exposed to dangers which attacked them from overhead, in the way in which small rodents are now attacked by carnivorous birds. The development of conjugate movements in the lateral plane followed later, with the shrinkage of the snout and the translation of the eyes forward in the head.
>
> As ontogeny is a condensed recapitulation of phylogeny, it is interesting to note that in the human embryo the optic vesicles when first formed are directly opposite to one another, from which position they gradually turn forward, so that at the third month of fetal life the optic axes of each eye diverge 45 degrees from the middle line; before birth they become parallel.
>
> Though nearly all mammals have, as in man, four recti and two oblique muscles connected with the eyeballs, the movements of the globes in all those below monkeys are comparatively slight.

I have chosen these citations of Collins' as of importance in support of my theory that voluntary unilateral ocular movement in man may be a reversal phylogenetically to an earlier type. There is further evidence to be adduced.

The phylogenetic development of ocular movement has been studied also by John I. Hunter.[10] He refers to the work of Elliot Smith, which

9. Collins: Arboreal Life and the Evolution of the Human Eye, Bowman Lecture.

10. Hunter, J. I.: Brain **46**:38, 1923.

showed the enhanced power of vision which developed as a result of the adoption of the arboreal life. Among the *Prosimiæ*, the reduction of the size of the nose in one group, the *Tarsioidea*, allows the eyes to come to the front of the face so that the fields of vision overlap. The enormous enhancement of the importance of vision which is thus effected leads to the sudden expansion of the cortical area for vision and its further specialization in structure.

Brouwer has found that a relation exists between the development of the large-celled nucleus of Perlia and the degree of approximation of the two eyes to one another. His conclusion is that these cells appear phylogenetically when the position of the eyes in the head is such as to render convergence possible. This is presumptive evidence that this nucleus sends its axons to the internal recti muscles, which effect the convergence developed coincidently with the appearance of the nucleus.

Hunter has studied the oculomotor nuclei in *Tarsius spectrum* and *Nycticebus tardigradus* and found that they contain the central nucleus of Perlia, which is smaller than in *Anthropoidea*. He believes that this accords with the conclusion of Brouwer that the nucleus appears phylogenetically when the recession of the face allows the approximation and consequence convergence of the eyes.

CONCLUSIONS

My conclusions from these facts are as follows: It thus appears that in the scale of development from *Rodentia* to man there is a gradual development of binocular vision. Monocular vision in the hare is so great that the optical axis in each eye measures 85 degrees of divergence from the middle line, almost a right angle. Hares have no conjugate movement of the eyes, and no consensual response of the pupils to light. This is the extreme of monocular vision and monocular movement, and yet in them monocular movement is comparatively slight. With the adoption of the arboreal life, binocular vision and associated binocular movement became greater, so that in *Tarsioidea* the fields of vision overlap and convergence becomes possible. At this period of development, the central nucleus of Perlia appears in *Tarsius* and is smaller than in *Anthropoidea*, it appears phylogenetically with the development of convergence. The evidence for phylogenetic development is still shown in the human embryo, for the optic vesicles when first formed are directly opposite one another; at the third month of fetal life, the optic axes diverge 45 degrees from the middle line, and before birth they become parallel.

Bielschowsky states that the vertical voluntary unilateral movement of the eyes is much more uncommon (erheblich seltener) than lateral voluntary unilateral movement. One might find an explanation for this

in Collins' statement that conjugate movement of the eyes in the vertical plane are innate in the infant, but in the horizontal plane are only acquired during the first six months of life. It is a well recognized law that functions phylogenetically older are more persistent and more firmly established than those more recently acquired in phylogenetic development. In man, therefore, the conjugate vertical movements would be expected to be more persistent than the lateral movements, and voluntary vertical unilateral movements of the eye to be much rarer than the monocular lateral movements, as Bielschowsky says they are. The lateral monocular voluntary movements are exceedingly rare (ausserst selten), according to Kolen, for in man conjugate ocular movement has become so pronounced as to lead to the formation of Hering's law, which I believe can be proved at fault in rare instances.

Unilateral movement in the hare postulates a cortical center controlling this movement; it is reasonable to assume, therefore, that if it occurs in man there must also be a more distinctly monocular representation in the cerbral cortex than in the normal man. This is the idea I had in mind in my reply to the question put to me by Gould in 1907. This idea of a more specialized cortical center for separate movements of one eye is a reversion to a more primitive type phylogenetically and does not, so far as I can determine, interfere with Kolen's theory of these separate movements being conditioned reflexes, although his presentation of the subject needs elaboration.

It might be interesting to examine the nucleus of Perlia in my patient, if the opportunity should ever be offered, but as the man presents the evidence of epidemic encephalitis it is probable that his lack of convergence is a loss of power once possessed and not a failure of development. It is therefore probable that the nucleus of Perlia would not show great difference from that of a normal man. If the lack of convergence were congenital, it would be still another point in favor of reversion to an earlier type, and I have no positive evidence that it is not congenital. It must not be forgotten, however, that my patient seems to be the only one known to have had voluntary monocular lateral movement with absence of convergence. I do not believe the unilateral ocular movement in my patient is entirely unilateral cortical innervation, but only in part. I recognize in him the evidence of bilateral ocular innervation in the action of the pupils and in the change in accommodation in both eyes during lateral movement of only one eye. There is no need to assume that the reversion to a primitive type is more than partial. It is impossible for me to accept the view that in my patient the extreme outward movement of either eye with persistence of the other eye in the primary position is to be explained as the result of equal innervation of the two eyes. I cannot believe that the extreme outward

movement is to be explained by mere relaxation of the internal rectus, especially when this muscle does not contract sufficiently for convergence; nor do I believe that it is to be explained as a "drifting out" of the eye to the normal position of rest as a result of strabismus divergens. The voluntary outward movement is decidedly greater than the divergence in the position of rest, and is indicative of actual innervation. The appearance of fatigue in the external rectus is also an evidence of voluntary innervation. There is much more probability of a partial reversion to a primitive type than of a complete reversion in man. One might be tempted to think that the fact that the man is of the negro race predisposes him to such a reversion, but all the other cases reported in the literature occurred in white persons, and one must, therefore, not ignore this fact.

DISCUSSION

DR. LA SALLE ARCHAMBAULT, Albany, N. Y.: I have a colleague at the Albany Hospital who is able to do exactly the converse of Dr. Spiller's patient. This physician is able to fix either the right or the left eye in normal midplane and establish unilateral convergence of the other eye; in other words, he is able to keep his right eye directly forward and to rotate the left eye internally. Likewise, he can look directly forward, keeping his left eye in the midplane and rotating the right eye. He has normal convergence, normal lateral associated movements and normal movements of elevation and depression. This condition has been present as far back as he can remember, and he has amused us with his ocular antics on more than one occasion.

I have no explanation to offer for this unusual innervation, except that possibly from an early age he has learned, as the result of practice, to dissociate volitionally what normally is an associated movement.

CHRONIC SUBDURAL ACCUMULATIONS OF CEREBROSPINAL FLUID AFTER CRANIAL TRAUMA

REPORT OF A CASE*

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WITH INTRODUCTORY REMARKS BY CHARLES A. ELSBERG, M.D.

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INTRODUCTION

After a cranial trauma, blood, cerebrospinal fluid or a serous effusion may accumulate underneath the dura and may, after a shorter or longer latent period, give rise to symptoms of disturbed function of the brain. The clinical pictures presented by these patients are often characteristic and similar, but insufficient attention has been directed to the variety of the lesions that may be found in the subdural space.

1. There may be a collection of fluid or clotted blood underneath the dura. This variety is the chronic subdural hematoma recently described by Putnam and Cushing. The blood usually overlies one hemisphere, although it may be found on both sides, and results in more or less marked compression of part of the brain. The symptoms are promptly relieved after fluid blood has been evacuated or after large blood clots have been removed.

2. Turbid or blood stained cerebrospinal fluid or a serous exudate may be found under the dura mater over one or both cerebral hemispheres. This is the condition to which Payr gave the name of meningitis serosa sympathetica. In this variety, the lesion is essentially an inflammatory one and is secondary to an infection of or actual suppuration in the bones of the skull or in the tissue of the brain. Unless the primary lesion is relieved, the serous fluid may become frankly purulent and a diffuse meningitis may follow.

3. The subdural space over part of one hemisphere may contain air or air and fluid, with perhaps air in the substance of the brain, in the arachnoid cisterns and in the ventricles (traumatic pneumocephalus) as shown in figures 1 and 2. The symptoms may be like those of the other varieties, but the nature of the lesion is easily recognized on the roentgenogram.

4. The accumulation in the subdural space consists mainly or entirely of cerebrospinal fluid, which may be blood-stained or xanthochromic. To this condition Payr gave the name meningitis serosa traumatica, but the term is not a good one as there is no evidence of an inflammatory process of the meninges.

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5. If the condition is unrelieved by surgical therapy, the fluid underneath the dura—whether it consists mainly or entirely of cerebrospinal fluid or whether it is a true subdural serous exudate—may finally coagulate and form a more or less solid mass over part of one cerebral

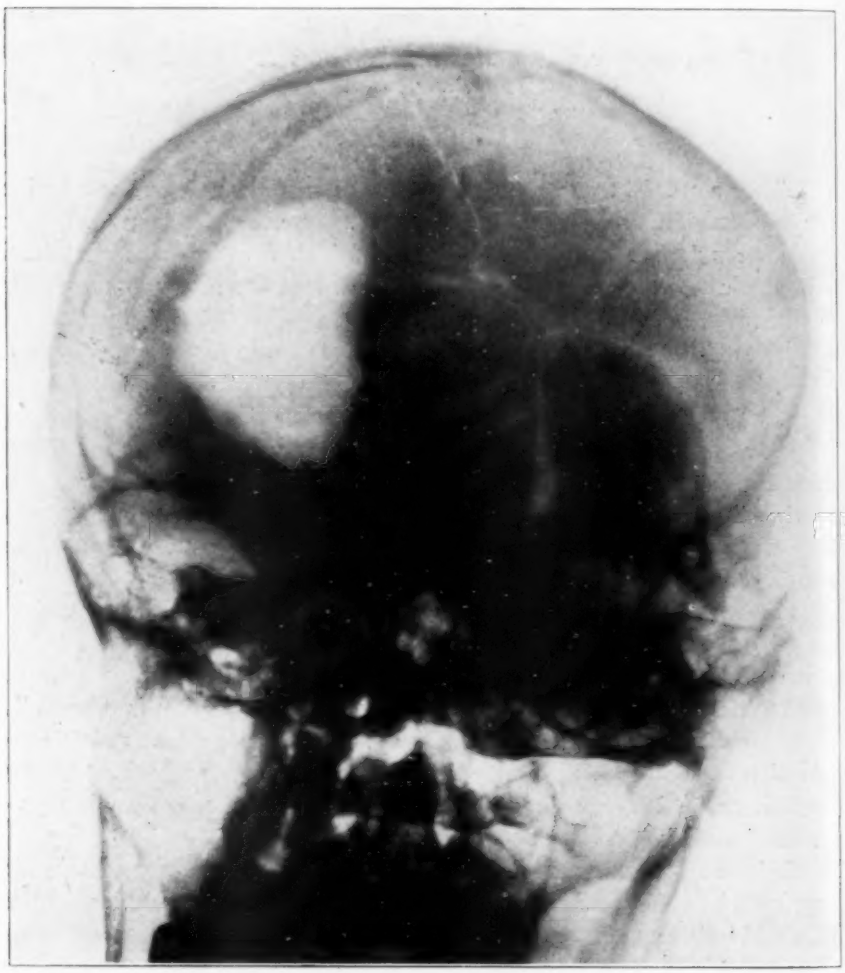

Fig. 1.—Anteroposterior view of skull of patient with traumatic pneumocephalus with a large collection of air underneath the dura, secondary to fracture of the skull.

hemisphere. This possibility was suggested by Dr. L. H. Cornwall, and he has been kind enough to call my attention to a brain in his collection at Columbia University College of Physicians and Surgeons, from which figures 3 and 4 were taken. They show a massive coagulum underneath the dura of one hemisphere. Sections of the coagulated mass showed,

microscopically, transparent tissue without any structure. There was no evidence of blood either in the coagulum or on the inner surface of the dura, and although markedly compressed, the cortex of the brain showed nothing abnormal. A somewhat similar case has been described by Abercrombie.[1]

Fig. 2.—Lateral view of patient shown in figure 1.

Subdural collections of cerebrospinal fluid after an apparently insignificant cranial trauma may cause symptoms so similar to those of chronic subdural hematoma that the distinction between the two conditions can be made only at the operating table. The subdural fluid may be blood-stained or xanthochromic; it may be localized over a small area

1. Abercrombie; Quoted by Schuberg in Arch. f. path. Anat. **16**:483, 1859.

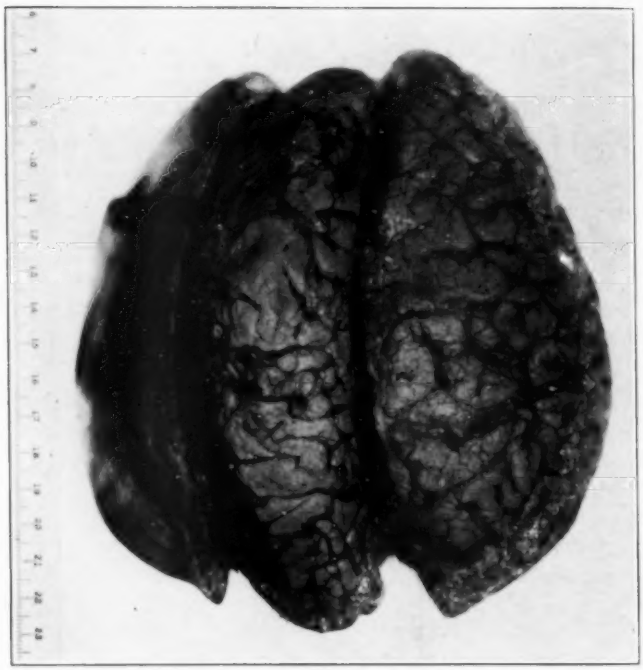

Fig. 3.—An old coagulated mass compressing the left hemisphere, the probable end-result of a subdural effusion of serum and cerebrospinal fluid. (From the collection of Dr. L. H. Cornwall at the College of Physicians and Surgeons).

Fig. 4.—The same brain as shown in figure 3. The subdural coagulated mass has been separated from the flattened hemisphere. (From the collection of Dr. L. H. Cornwall at the College of Physicians and Surgeons).

or it may compress a large part of one cerebral hemisphere. Aside from the presence of the large collection of fluid, of a thin fibrinous membrane on the inner side of the dura and of the compressed brain, there are no other gross pathologic changes. The cortex of the compressed brain is normal in appearance, without any evidence of blood clot or fibrin on its surface.

In these patients, the effect of the injury of the head must have been to cause a rupture of the arachnoid in an unknown location, with the escape of cerebrospinal fluid into the subdural space. It is not clear why the fluid should collect outside the arachnoid; there is probably a valvelike action at the site of the torn arachnoid, so that cerebrospinal fluid can escape into the subdural space but cannot reenter the subarachnoid spaces from which it could be absorbed. The mechanism on which the accumulation of fluid is based is probably similar to that which occurs in traumatic pneumocephalus with chronic subdural and subarachnoid collections of air.

In contradistinction to the subdural hematomas, which are permanently remedied by the evacuation of blood or the removal of blood clot, the chronic subdural collections of cerebrospinal fluid have a great tendency to reaccumulate. If the fluid is emptied by aspiration through a trephine opening in the skull or by a more extensive surgical procedure, it reaccumulates within a short time, and repeated removals of fluid may be necessary—as in the case reported in this paper. The constant reaccumulation of fluid finally causes a marked deformation of the brain which may persist over a long period, so that the brain does not show a tendency to expand and fill the cranial cavity in the characteristic manner seen after the removal of massive blood clots or of meningeal new growths.

In the following case, a diagnosis of chronic subdural hematoma led to the operative intervention and to the recognition of the condition as one essentially different from that described by Putnam and Cushing. The case is remarkable, not only on account of the constant reaccumulation of the fluid and the final relief of symptoms after an added subtemporal decompression, but also because of the later paucity of evidence of neurologic disturbances. That the compression of so large a part of the left cerebral hemisphere in a right-handed person could occur with only temporary and relatively slight disturbances of speech and of motor power is remarkable, and is additional evidence of the extent to which the brain can accommodate itself to changed relations and of the degree of substitution of functions of which the cerebral hemispheres are capable.

SUBDURAL ACCUMULATIONS OF CEREBROSPINAL FLUID

The subject of subdural accumulation of blood or fluid after a cranial trauma has, during the past few years, attracted considerable interest, and several important papers on the subject have been published. In 1924, Naffziger [2] called attention to the occurrence, after injuries to the skull, of collections of fluid between the dura and the arachnoid, and described the clinical symptoms that he observed. The subdural accumulation of fluid occurred within a few hours or days after the cranial trauma. At operation, the fluid was found to be either clear or blood stained. Naffziger considered that the fluid was normal or bloody cerebrospinal fluid that had escaped as a result of a tear in the arachnoid, and had collected there because it could not be absorbed from the subdural space. The condition was relieved by evacuation of the fluid and drainage through a subtemporal decompressive opening.

Putnam and Cushing,[3] under the title of "Chronic Subdural Hematoma," made a careful study of an interesting group of cases of which many isolated instances had been reported in medical literature. In these cases, weeks or months after a cranial trauma had taken place, symptoms of a cerebral disturbance developed, and at the operative intervention a large amount of partly organized blood clot or fluid blood was found under the dura. These authors made a careful study of the pathologic condition of the changes that were encountered and gave a useful summary of the symptoms of chronic subdural hematoma and the surgical methods for its relief. In their paper, the literature of the entire subject was collected, and the various theories that had been proposed to explain the condition were subjected to a critical analysis.

They considered also the relation of chronic subdural hematoma to the so-called pachymeningitis interna hemorrhagica, to which Trotter [4] had called attention in a paper published in 1914.

Subdural effusions or accumulation of fluid had also been described by Payr,[5] who believed that the condition was due to a serous meningitis. He divided his cases into two groups—"meningitis serosa traumatica," which appears early and is due directly to the trauma, and "meningitis serosa sympathetica," in which the protein content of the fluid is high and the end-stage of which is a true meningitis. In one

2. Naffziger, H. C.: Subdural Fluid Accumulations Following Head Injuries, J. A. M. A. **82**:751 (May 31) 1924.

3. Putnam, T. J., and Cushing, Harvey: Chronic Subdural Hematoma, Arch. Surg. **11**:3 (Sept.) 1925.

4. Trotter, W.: Chronic Subdural Hemorrhage of Traumatic Origin and its Relation to Pachymeningitis Hemorrhagica Interna, Brit. J. Surg. **2**:271, 1914.

5. Payr, E.: Meningitis Serosa bei und nach Schädelverletzungen, Med. Klin. **12**:841 (Aug. 6) 1916.

of Payr's cases, an operation, performed ten days after a slight trauma to the frontal region, disclosed that the subdural space contained about a half liter of clear fluid, which so greatly compressed the apparently uninjured hemisphere that a considerable portion of the falx was exposed.

REPORT OF CASE

The following case that came under observation is reported as a contribution to the subject of subdural collections of cerebrospinal fluid which may follow a slight cranial trauma.

History.—D. R., a furrier, aged 20, was referred by Dr. E. D. Friedman and was admitted to the neurological service of Mount Sinai Hospital on April 26, 1925. His birth was normal, and he always had been well up to the present illness. He was right-handed and had been known as a "strong man", able to bend iron rods and to break heavy iron chains. Six weeks prior to admission, during a boxing bout, he was struck on the bridge of the nose and was stunned for a minute but did not lose consciousness. The following morning he suffered from headache, which was severe enough to prevent him from doing his usual work. The headache continued for two weeks, during which he had several attacks of vomiting. In another hospital, two lumbar punctures had been performed; the first was followed by temporary relief of the symptoms, and the second was without benefit. The fluid removed was xanthochromic. Four days prior to admission to Mount Sinai Hospital he felt dizzy and weak and had diplopia, and on the following day had a generalized convulsion. On the day of admission, he developed a marked disturbance in speech.

Physical Examination.—The man looked strong, was well developed, and did not appear ill. He understood commands and obeyed them promptly, but he had no spontaneous speech. His answers were limited to "yes," "all right" and a few similar short expressions. He was able to read, but could write only the first two letters of his name.

Cranial Nerves: Acuity of vision was normal and the fields were complete; there was a beginning papilledema in both eyes. The pupils were equal and reacted promptly to light, direct and consensual, and in accommodation. Movements of the eyeballs were good in all directions; there was no nystagmus. Weakness of the right side of the face, supranuclear in type, was present.

Motor Disturbances: There was no rigidity of the neck nor Kernig sign. There was slight but distinct weakness of the right lower extremity. The tendon reflexes of both upper and lower limbs were somewhat hyperactive and everywhere were equal on the two sides, with the exception of the right achilles reflex, which was more active than the left. Coordination was undisturbed in all the extremities. Only the left lower abdominal reflex could be elicited; the cremasteric reflexes were active; there was a distinct Babinski extensor response on scratching the sole of the right foot.

Sensation: No sensory disturbance of any kind, either of the cutaneous or of the deep sensibilities, was present. Cotton and pin prick were well felt all over the body, as was the vibration of a tuning fork. The muscle-joint-tendon and sterognostic sense were not disturbed. The temperature was 98.6 F.; the pulse rate 72. General physical examination showed nothing abnormal.

Course.—On April 27 (the day after admission), the aphasia and the agraphia had entirely disappeared, and no evidence could be found of the disturbance of speech that had occurred the preceding day.

On April 30, the pulse rate was from 46 to 64. Lumbar puncture showed xanthochromic fluid under greatly increased pressure. The fluid contained 12 lymphocytes to the cubic millimeter.

On May 5, the general condition was unchanged, but for several days the patient had complained of diplopia. The right palpebral aperture was slightly larger than the left. Bilateral external rectus weakness and a fine horizontal nystagmus were present. The tongue deviated slightly to the right. Grip with the left hand was stronger than with the right. A Babinski response was sometimes elicited on the right and sometimes on the left side. The papilledema had advanced, so that now there was a swelling of 3 diopters in each eye, with small hemorrhages.

On May 12, the symptoms and signs had remained the same for a week except that the neck had become somewhat rigid and the papilledema had increased to 4 diopters. The patient was fully conscious and complained of continuous headache and of occasional diplopia. Lumbar puncture showed the same xanthochromia and high pressure; the fluid contained 70 cells to the cubic millimeter, 96 per cent of which were lymphocytes. The Wassermann reaction with both the blood and the spinal fluid was negative; the blood count and blood pressure were normal. Roentgenographic examination of the skull was negative.

Operation.—The diagnosis of a large subdural effusion of blood after a cranial trauma was made, and evacuation of the fluid was decided on. A vertical incision 3 cm. long was made over the upper part of the left frontal region, at least 5 cm. outside the midline and just behind the coronal suture, and a button of bone removed with a trephine. The exposed dura was tense and bluish. A small incision was made in the dura, and the inner surface was found to be covered by a fine membrane, no thicker than tissue paper, which had the appearance of a fine blood clot. As soon as this membrane was opened, a large quantity of brownish yellow fluid spurted out under pressure. A catheter was inserted through the opening and between 350 and 500 cc. of fluid was removed by aspiration. The cavity was then explored with a large probe, which could be passed directly toward the median line for a distance of 5 cm. before being arrested. It passed forward to the frontal bone and backward almost to the occipital region, so that a large subdural cavity must have resulted from the compression of the frontal and temporal lobes. The cavity was thoroughly washed out with saline solution, the dural incision sutured and the scalp closed without drainage. The fluid withdrawn was chrome yellow and contained many well preserved red blood cells and a few hematin crystals. At the close of the operation, the patient stated that he no longer saw double.

Postoperative Course.—Two days after the evacuation of the fluid, headache and diplopia returned. The disks were still swollen to 4 diopters, but there was no rigidity of the neck nor Kernig sign, no weakness of any extremity, no hyperactivity of the tendon reflexes and no Babinski sign. A needle was introduced through the former trephine opening and more than 200 cc. of fluid allowed to escape. The headache and diplopia persisted, however, and there was no recession of the papilledema, so that it was considered that perhaps there was a similar collection of fluid on the right side. To exclude this possibility a button of bone was removed over the right frontal region, and a small incision made in the exposed dura. No more than 1 cc. of xanthochromic fluid could be evacuated from beneath the dura. The tension of the brain was not increased, but was somewhat below normal. The left side was then aspirated through the old trephine opening, and 180 cc. of yellow fluid evacuated. In order to obtain roentgenograms of the cavity, 60 cc. of air was injected (figs. 5 and 6).

At each of the aspirations of the left frontal region, the needle could be passed for a considerable distance in several directions before the resistance of brain tissue was encountered. The question arose whether there was a lamellated blood clot which prevented the cortex from expanding to fill up the large space from which the xanthochromic fluid had been withdrawn. An exploratory operation was therefore decided on and was performed by Dr. Elsberg on May 26.

Fig. 5.—Anteroposterior view of skull of patient after injection of air into subdural space, showing the large cavity. In this figure and figure 6, the entire cavity is not filled with air.

Second Operation.—Under general anesthesia, a large frontoparietal flap was made and turned down on the left side. Through a small incision in the dura, which was found to be tense, about 300 cc. of yellow fluid was evacuated. The dural sac was then widely opened and the condition illustrated in figures 7 and 8 was seen. There was no evidence of a blood clot anywhere, except a thin layer

of reddish material on the inner surface of the dura. The cortex appeared normal in color and the blood vessels were of normal size, but the frontal, temporal and parietal lobes were much displaced or compressed, so that the floor of the entire anterior fossa as well as of the greater part of the middle fossa was exposed. The falx cerebri was visible in the median line, and the optic nerve, the ophthalmic vein, and the third nerve were seen on the floor of the skull. During the exploration the exposed brain did not change its position, and nothing was found to explain the fact that the brain showed no tendency to

Fig. 6.—Lateral view of patient after injection of air into subdural space. The similarity of this figure and figure 5 to the appearance of the case of traumatic pneumocephalus (figs. 1 and 2) should be noted.

expand and fill the large empty space. When the dura was closed, the cortex lay in exactly the position in which it had been when the dura was opened. The bone flap was returned into place and the scalp wound closed.

Postoperative Course.—On the day after the operation a careful, complete examination of the patient was made. The motor power was good in all limbs, and grip with the right hand was stronger than the left. The visual fields were

complete without the smallest defect. Speech was absolutely normal. The movements of the right side of the face were as good as the left. The reflexes in the right upper and lower extremities were no more active than those on the left. Tactile and pain sensibility, as well as muscle-joint-tendon and vibratory sense were normal all over the body and the limbs. There was no astereognosis.

On May 29, (four days after the operation), the swelling of the disks was 4 diopters in each eye.

On June 5, the left subdural space was punctured, and 240 cc. of yellow fluid was removed.

On June 9, 270 cc. of yellow fluid was removed; during the aspiration, the patient complained of headache.

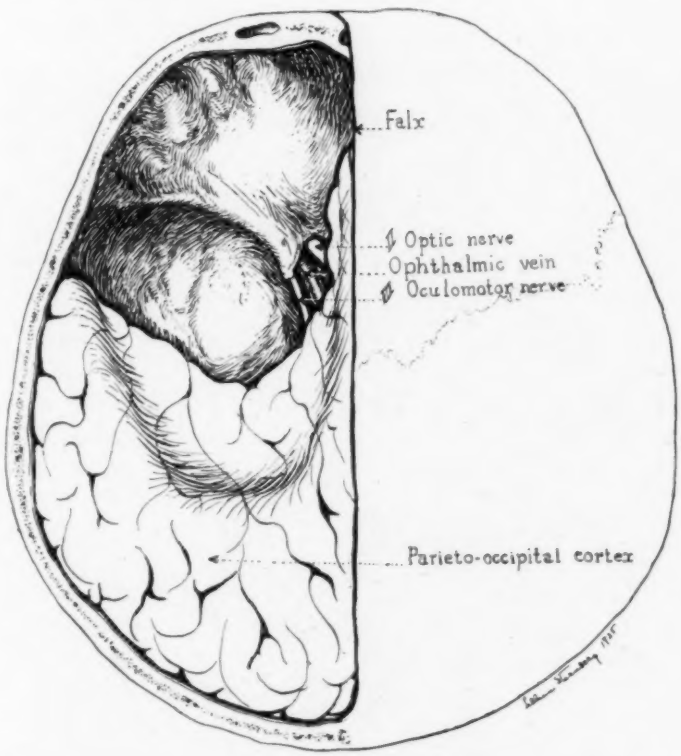

Fig. 7.—Diagrammatic illustration showing the compressed brain seen at operation, with the floor of the anterior and of part of the middle fossa exposed and the optic nerve, ophthalmic vein and oculomotor nerve visible.

On June 18, a similar amount of fluid, which was lighter in color and under less tension than previously, was removed.

The end of June found the patient about as before. Subjectively, there were headaches and transient blurring of the vision. Objectively, the only constant symptom was swelling of 4 diopters in each eye, with a visual acuity of right eye, 20/40; left eye, 20/50.

On July 9, the general condition and the symptoms were unchanged. By aspiration, 200 cc. of fluid was removed, the color being lighter than at any

previous aspiration. After the first 90 cc. had been removed, further suction on the syringe caused headache referred to the occipital region.

On July 22, the swelling of the right disk was 5 diopters. Drainage of the left subdural space was considered but was rejected because of the danger of an infection of the meninges. In the effort to benefit the papilledema a right subtemporal decompression was decided on, and I performed the operation under local anesthesia on July 24. The dura was tense and bulging, and there was a slight increase of fluid beneath it. The surface of the brain was normal. It

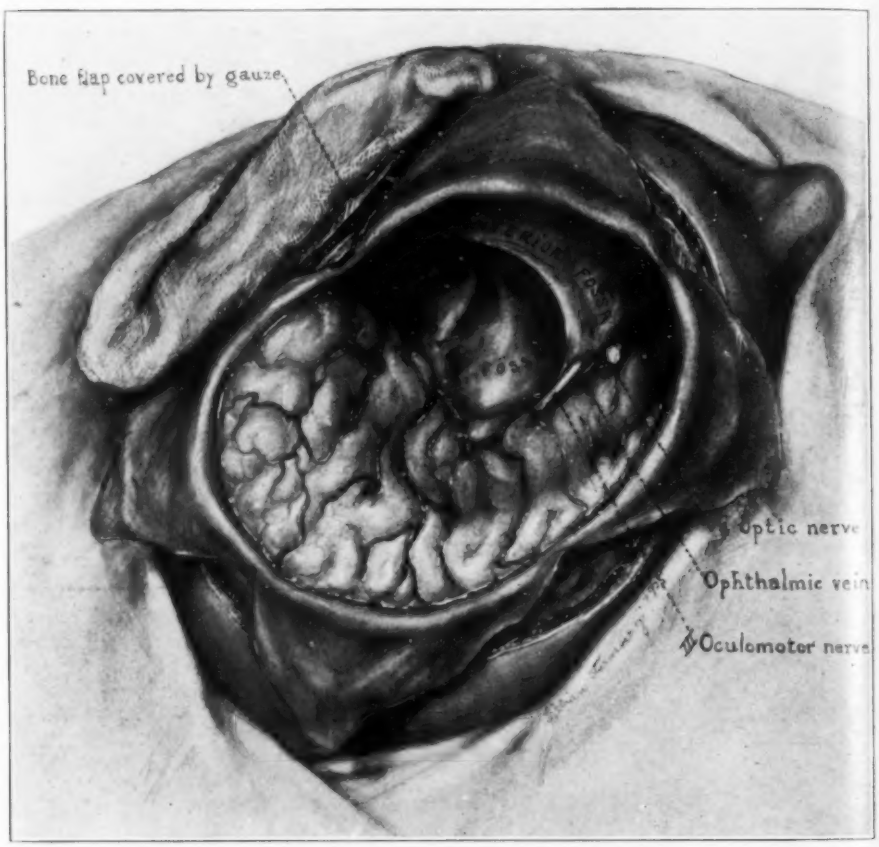

Fig. 8.—Condition found at operation after the osteoplastic flap had been turned down and the dura widely opened.

filled the subdural space and bulged only slightly through the decompressive opening. Following the operation, 200 cc. of yellow fluid was removed from the left side by puncture and aspiration; exploration with the needle showed that the subdural cavity was about as large as it was at the time of the osteoplastic exposure.

Two weeks later the swelling of the disks had receded to 1 diopter, and the vision had improved to 20/30 in each eye. There was slight bulging at the site of the decompression.

On August 20, 250 cc. of fluid was removed by puncture and aspiration of the left subdural space. While the fluid was being removed, the patient complained of pain referred to the retro-orbital and occipital regions. The headache was relieved, however, when the fluid removed was replaced by air.

Excepting for the periods following the operative procedures and the days following the aspirations, the patient had been up and about in the ward for several months. With the exception of the receding papilledema, there were no longer any signs of disturbed function. He was discharged from the hospital on August 22.

The patient was seen in the Follow-Up Clinic at intervals up to eighteen months after discharge. He had no headache, and the fundi were normal. Physical examination showed normal reflexes and normal motor power in all the limbs.

COMMENT

In all the cases of so-called chronic subdural hematoma, the fluid was enclosed between a more or less thick lamellated clot on the dural side and a thin fibrous layer overlying the arachnoid. In our patient, there was only a thin layer or membrane on the inner surface of the dura, while the arachnoid and the underlying cortex appeared normal. The membrane on the inner side of the dura was so thin that it could not have been removed, and the microscopic examination of a small fragment of excised dura failed to show any evidence of a distinct layer of blood clot on its inner surface.

There must have been, of course, in some location a communication between the subdural and the subarachnoid spaces, for the fluid removed by lumbar puncture was xanthochromic.

An interesting feature of this case is that the extreme compression of the left hemisphere caused so few symptoms and signs of disturbed function, and that most of the changes in the reflexes and the cranial nerves disappeared quickly—long before the condition had been relieved by surgical intervention. The entire frontal lobe as well as the motor, speech and sensory cortex were crowded into a space less than a fourth of what it should normally occupy. An aphasia of less than twenty-four hours' duration and a transitory weakness of the right arm were the main results of this extensive change.

The compression (if compression it was) developed during a period of six weeks and involved the left side of the brain in a right-handed person. In the case of, for example, a dural tumor in the same location, the compression would be far more gradual, but a tumor as large as the accumulation of fluid in this case would have certainly caused more signs of cerebral disturbance. The pressure from these accumulations of fluid is therefore less apt to give marked signs of interference with the functions of even important areas of the brain—a fact to which reference has been made by other authors. Notwithstanding this, however, it is remarkable that there were not, in this patient, persistent dis-

turbances of speech, changes in the visual fields and marked motor and distinct cortical sensory loss.

There is no satisfactory explanation for the failure of the brain to expand after the removal of the fluid during the wide osteoplastic exposure of the left cerebral hemisphere, and the repeated evacuation of the collection of fluid by aspiration. There was neither clot nor membrane to prevent the expansion, and I should have expected the brain to fill in the defect just as one observes the compressed brain quickly fill the space from which a large endothelioma has been removed. Trotter has observed this lack of expansion after the removal of the blood clot in two of his four patients with chronic subdural hemorrhage; Payr describes a similar condition in one of his cases of "meningitis serosa," and so does Mayo [6] in a reported case. The possibility of a congenital anomaly of the hemisphere with a superimposed collection of subdural fluid caused by the trauma would have to be considered as a possibility, but in the patient reported, there was no evidence in the history to support such a theory.

Of equal interest is the constant reaccumulation of the fluid. On its first removal it was a deep brown, with well preserved red blood cells. Three months later it was slightly yellow. The amount that reaccumulated remained about the same—more than 200 cc.—which was removed repeatedly. Although, unfortunately, no careful chemical examination of the fluid was made, it is probable that it was cerebrospinal fluid that entered the subdural space through a tear of the arachnoid, either somewhere over the convexity of the hemispheres or over one of the basal cisterns.

The recession of the papilledema after a right subtemporal decompression, in spite of the persistent reaccumulation of fluid over the left hemisphere, is of interest. The tension of the right temporal lobe, at the time of the decompressive operation, was not great, and the bulging in the subtemporal region was never marked. The swelling of the disks promptly subsided after the operation, and the disks have remained flat up to the last examination. As the patient feels perfectly well, he has refused to permit another lumbar puncture or further aspiration to determine if there is still fluid over the left side of the brain.

Finally, the relation of headache to the negative pressure caused by removal of the fluid is of interest. During the course of evacuation of fluid from the ventricular system under local anesthesia and while air was being injected into the ventricles for the purpose of pneumog-

6. Mayo, C. H.: A Brain Cyst—the Result of Injury, Causing Aphasia, Hemiplegia, etc., Evacuation, Complete Recovery, New York M. J. **59**:434 (April 7) 1894.

raphy, it was often observed that the patient complained of headache after the first 10 or 20 cc. of fluid had been removed. The headache was especially apt to occur if the fluid was allowed to escape rapidly or was actually removed by aspiration and when air was injected too quickly. The headache was localized in the retro-orbital region or was more diffuse, and the pain due to the removal of fluid was often promptly relieved by the injection of air. In the patient whose history has been recorded, the removal of fluid from the subdural space caused intense headache often spoken of as "behind the eyes." One is accustomed to associate headache with increased intracranial pressure, but it may occur also from a sudden diminution of pressure. It seems probable, therefore, that in some cases headache may be due to a variation in intraventricular pressure and, as has been pointed out elsewhere, may be, in many instances, due to a change in the conditions of pressure within the third ventricle. On the other hand, an increase or decrease of general intracranial pressure may have the same effect.

The more or less generally accepted theory that headache is due to distention of the dura seems to be no longer tenable, not only because the dura itself is insensitive but because headache can be produced by variations in intraventricular pressure even when the dura is widely open. It is undecided, however, whether changes in the tension of some localized area of dura may indirectly play a part.

SUMMARY

A previously healthy young man, six weeks after a slight cranial trauma, developed signs of a left cerebral lesion, with increasing papilledema. Operations disclosed a large subdural collection of xanthochromic fluid, a left hemisphere compressed to less than a fourth of its normal size and a small amount of fluid of the same character beneath the dura of the right hemisphere. During a four months' period, the left subdural space was more or less completely emptied nine times and more than 2,300 cc. of fluid was removed. Except for headache and papilledema, all signs of disturbed function soon disappeared. The headache and choked disks were relieved only after a right subtemporal decompression.

TUMORS OF THE PINEAL GLAND*

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In this paper two cases of tumor of the pineal gland are reported, and an endeavor has been made to summarize all such cases in the literature in order to determine the most frequent signs and symptoms. The most comprehensive summaries at present in the literature are those of Bailey and Jelliffe, in 1911, who reviewed fifty-nine cases, and of Seigneur, in 1912, who described sixty-five tumors of the pineal gland. In the present paper 113 cases are tabulated with respect to microscopic picture, symptoms and incidence according to sex and age. Valuable assistance in the preparation of this report has been rendered by Dr. G. Y. Rusk, who furnished the pathologic material, and by Dr. H. C. Naffziger, from whom the clinical data on the first case were obtained.

REPORT OF CASES

CASE 1.—*History.*—The patient, a white woman, aged 49, entered the neurosurgical service of the University of California Hospital in February, 1920, complaining of blindness, left frontal headache, forgetfulness and incontinence of urine and feces. The father had died of paralysis, but the family history otherwise was without significance. The patient had had measles, mumps, chickenpox and scarlet fever in childhood, and had been troubled by frequent headaches all her life. The only accident was a fall from a stepladder more than twenty-five years before admission; she had had two operations, one for a retroverted uterus in 1905 and one for excision of several cysts of the cervix in 1914. She had suffered from incontinence of the bladder and bowels since 1917. The menopause occurred in February, 1919, one year before entry into the hospital.

The present illness dated from 1914, when she became exceedingly nervous. Her head felt "as if it would fly off," and there were tight pressure pains in the back of the head, which were worse in the morning and bore no relation to meals. The patient's vision began to fail in February, 1914, and she thought her memory became less active. Once, in December, 1915, she became dizzy and fell to the floor. A second attack occurred three weeks later. During these attacks the patient experienced no emesis or headache, but the lips became pale and she seemed to lean forward and sink downward, usually falling toward the left side.

During 1916, the impairment of vision increased and she experienced a stinging pain in the right eye in the morning; the pain was greatly relieved by glasses. From June to October, 1916, she heard what appeared to be pulsating waves, and a sweet odor (as of chocolate) was frequently noticed.

In August, 1916, the attacks of dizziness became worse and she frequently bumped into objects on her left side and seemed to fall toward the left. On Aug. 8, 1916, she entered St. Luke's Hospital with the following complaints: (1) tight vise-like pains in the back of the head, which occurred about every

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third day; (2) a feeling in the spine as though it contained an iron rod, and (3) failing vision. Examination of the eyes at that time showed pupils slightly irregular, the right smaller than the left, slight reaction to light, but normal reaction in accommodation. The condition of the eyes was diagnosed as retrobulbar neuritis. The visual fields showed irregular contraction for colors. The patient left St. Luke's Hospital on Aug. 16, 1916, with a final diagnosis of "multiple sclerosis (?)."

During the fall of 1916 and spring of 1917, the patient's vision failed steadily until she became completely blind. The eyes tended to turn to the extreme right or left. The reflexes were greatly increased, and she kept nervously wringing her hands and twiddling her fingers. Convulsive seizures became frequent and severe in the spring and summer of 1919. From 1916 to 1919, the patient could not walk and attempts resulted in falls. She was mentally bright at times but was usually drowsy. Visitors tired her and increased the frequency of convulsions. The patient was entirely disoriented and was ignorant of the blindness, which she attributed to the darkness of the room. Many visual hallucinations were experienced. She held many false ideas, and a prominent symptom was a habit of stopping in the middle of a sentence and forgetting that she had been talking.

For a week in December, 1919, the patient had been troublesome to attendants because of restlessness and frequent falls. On Dec. 20, 1919, a copious flow of clear fluid from the left nostril was followed by a spectacular and rapid recovery, which appeared complete except for persistent blindness and amnesia covering the preceding three or five years. On Jan. 15, 1920, the roentgen ray showed complete obliteration of the outlines of the sella turcica. Drainage of fluid from the nose stopped on February 14, and was followed within twenty-four hours by some return of the former symptoms of forgetfulness, left frontal headache, positive Romberg sign, loss of coordination, inability to feed herself, and incontinence of urine and feces. The patient entered the University of California Hospital on Feb. 15, 1920.

Physical Examination.—The patient was stout; blindness, with slight exophthalmos, was present. She was able to sit and stand alone and appeared mentally clear, oriented and able to give her past history up to 1915. Examination of the chest, abdomen and spine gave essentially negative results. The urine was normal; the blood contained 7,000 white cells; the blood pressure was 120 systolic; 80, diastolic.

Neurologic Examination (Cranial Nerves).—The first nerve showed anosmia at the time of examination, with hallucination of a sweet odor at times in 1916, but none since then. The second nerve showed scotoma in 1916, and visual hallucinations and failing vision from 1914 to complete blindness in 1917. Examination of the fundi showed secondary optic atrophy. The third, fourth and sixth nerves did not show any evidence of diplopia. The patient presented ptosis of both lids, more marked on the right, and a slight exophthalmos, which was greater on the left. The pupils were irregular, the right being slightly larger, and showed no reaction to light or in accommodation. The eyes tended to turn downward when the patient was asked to look to the right or left, and would turn to the right and up when she was asked to look up. Lateral nystagmoid movements were seen when the patient tried to look to the right or left. The patient could converge fairly well, and showed no strabismus. The fifth nerve showed occasional numbness of the right side of the face, and three attacks of flushing had occurred on the right side of the head and face during the two weeks before entry. There was diminution to touch and pain over the orbital branch of the

fifth nerve, although no change in temperature and deep sense was noted. The motor and corneal reflexes were normal. Taste was absent until Dec. 20, 1919, when a large amount of clear fluid escaped from the left nostril; since then taste had been present, though slightly diminished, over the anterior two thirds of the tongue. The seventh, eighth, ninth, tenth and eleventh cranial nerves were normal, both subjectively and objectively. Examination of the twelfth nerve showed that the tongue protruded in the midline but if held out for any time repeatedly turned to the right. A fine tremor was seen, although no marked fibrillations were present.

Examination of the extremities showed them to be altogether normal as to both motor and sensory functions. All normal and no pathologic reflexes were present. There was no ataxia, asynergia, or adiadokokinesis, and the finger-to-nose, heel-to-knee, and finger-touching tests were well performed.

Summary.—Localizing symptoms grouped together showed: cerebellar symptoms: in 1916, the patient tended to fall to the left, although at the time of examination she did not show a tendency to fall toward either side. Giddiness was present only when she attempted to stand after lying down. A complaint was made of pain high in the back of the neck, but no cervical rigidity was present. Regarding the frontal lobe, there was an amnesia of from three to five years' duration up to Dec. 20, 1919. There had been no signs of motor irritability or of paralysis. The only sensory disturbance was a diminution to touch and pain over the orbital branch of the fifth nerve. A postneuritic optic atrophy, progressing to blindness, was present. Examination of the vestibular apparatus showed that it was intact but hyperactive.

Course.—Following the patient's entrance into the hospital, the cerebrospinal fluid continued to flow freely from the left nostril. A right subtemporal decompressive operation was performed by Dr. Howard C. Naffziger on Feb. 24, 1920, and a moderate intracranial tension and a moderate amount of cerebrospinal fluid were found. On March 10, there was increasing motor involvement of the left facial area with increased spasticity and a positive Kernig sign but no rigidity of the neck. Forty cubic centimeters of clear spinal fluid was withdrawn. The urine contained many pus cells, and on culture *Streptococcus viridans* was found.

A right temporofrontal exploration was made by Dr. Naffziger on April 16, 1920. By making a frontoparietal bone flap, opening the dura and elevating the frontal lobe it was possible to secure an exposure of the floor of the anterior fossa, the anterior clinoid process, the anterior margin of the sella and the infundibulum. The normal anatomic relation had been destroyed by a large, bulging, cyst-like accumulation which proved to be a dilated infundibulum. This was opened and the ventricular cavities could be seen within. A small portion of the anterior wall was removed with the hope of establishing additional drainage tracts. The condition indicated an obstructive hydrocephalus from a lesion above the hypophysis. The dura was closed, the bone flap and bone buttons were replaced, and the scalp was closed.

Microscopic examination of the tissue removed showed it to be composed largely of glial fibers with scanty glial nuclei. The general picture was that of tissue from the tuber cinereum.

On May 11, a prominent, soft, fluctuating swelling appeared at the hinge of the operative flap. Spinal puncture on May 25 obtained clear fluid under decreased pressure. The white blood count was 32,000. The patient became progressively more stuporous after May 9, and died on May 27, 1920.

Postmortem Examination.—The autopsy was limited to an examination of the brain. The scalp showed a well healed surgical incision in the right frontoparietal region. The skull separated normally, except for adhesions at the site

of the decompression, and was unusually thick and heavy, especially posteriorly, where a dense outer table reached about 0.5 cm. in thickness. The diploe generally was dense. The inner surface of the skull cap showed a number of abnormally broad and deep depressions, especially along the midline at the site of the pacchionian granulations. The under surface of the dura, over the vertex in general, was normal. The convolutions of the right hemisphere showed slight flattening around the site of the decompression. Connecting the dura with the brain surface adjacent to the longitudinal fissure were a number of fine thread-like cords, some of which contained blood. When the brain was raised, adhesions were found about the site of the decompression and under the frontal region.

The brain separated from the dura in the region of the cribriform plate with difficulty, owing to the formation of a number of cerebral hernias in this region, and there was a distinct opening from the left lateral ventricle through the dura. The sella was enlarged in all directions, the posterior region being completely absent. A hole was present in the tuber cinereum, and a number of delicate

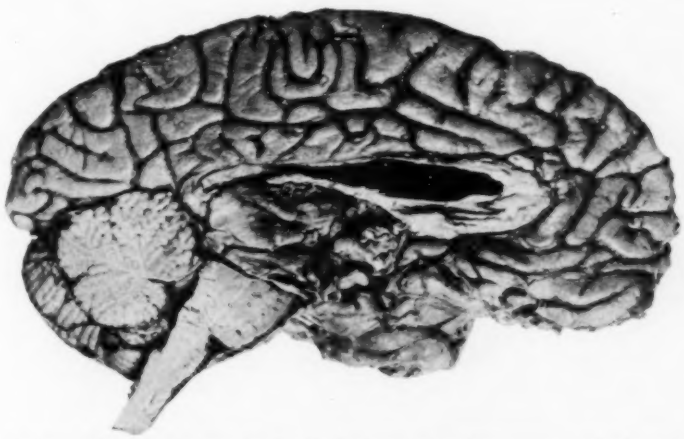

Fig. 1 (case 1).—Brain cut in sagittal plane, showing tumor of pineal gland.

adhesions could be seen around this general region. The hypophysis was pale, and its tissues were recognized with difficulty. A number of additional herniations were seen over both temporal lobes anteriorly and a few over the occipital lobe.

On removal of the brain, the first nerves appeared slightly thin, the bulbs tearing off in the region of the cribriform plate. The optic nerve was markedly thinned out, lying in the walls of the tuber cinereum. Through the hole in the tuber cinereum, the third ventricle was seen to be dilated. The anterior and middle commissures were stretched out. Just behind the middle commissure in the superior posterior portion of the third ventricle was a slightly reddish, smooth, tumor mass, spherical in outline, about 1 cm. in diameter. The pia between the frontal lobes and around the sylvian fissures was somewhat tough and thickened, that extending around the isthmus to the region of the straight sinus being particularly tough.

The convolutions on both sides of the brain appeared somewhat flattened, especially over the parietal and temporal regions and more pronounced on the right. There was a marked bulging of the right temporal region inferior to the posterior part of the sylvian fissure. This marked the site of the decompression operation. The inferior surface of both temporal lobes showed irregular lacera-

tions, probably produced in removing the brain, due to herniations through the dura. A slit appeared on the right ventral surface just anterior to the olfactory bulb. The cerebrospinal fluid doubtless escaped from the lateral ventricle into the nose through this opening.

When the brain was sectioned in the mid-sagittal line, an almond-shaped tumor was seen in the region normally occupied by the pineal gland. Its long diameter was anteroposterior and measured 37 mm. Its shortest diameter (vertical) was 17 mm., and its lateral diameter approximately 30 mm. in extent. A small, slit-

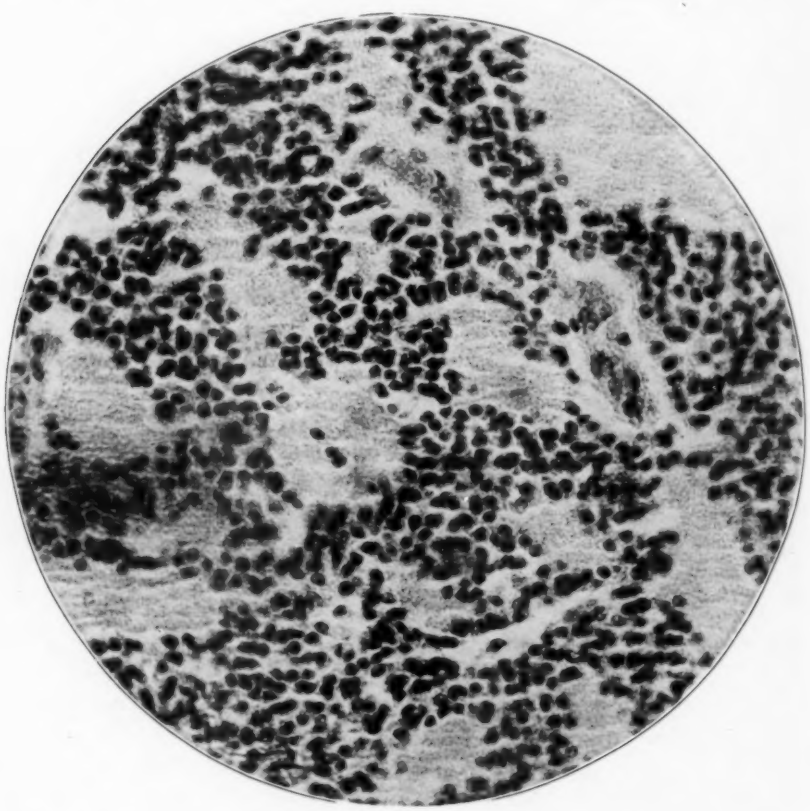

Fig. 2 (case 1).—Typical section through tumor showing granular areas and masses of cells. Hematoxylin and eosin stain; low magnification.

like cavity could be seen running longitudinally through the center of the tumor. The aqueduct of Sylvius was pressed shut but could be opened by raising the tumor. A ventral view showed the olfactory bulbs intact and the optic nerve, chiasma, and tracts present but somewhat thinner than normal. The occipitofrontal diameter in the temporal region was 14 cm. The cerebellum appeared normal, except that the vermis seemed somewhat compressed and the fourth ventricle slightly flattened.

Microscopic Examination.—On microscopic study, the tumor was seen to consist of masses of cells among which were compact, acellular, granular areas. These areas stained with eosin, were irregularly shaped, and comprised about

one third of the area of the section. The cells were irregular in shape and were almost completely filled with round or oval nuclei, from 7 to 12 microns in diameter. The nuclei contained deeply staining chromatin granules and nucleoli, and showed no definite arrangement. Mallory's phosphotungstic acid hematoxylin demonstrated numerous neuroglia fibrils within the cells and bridging the acellular masses. No mitoses, cysts, or hemorrhagic areas were seen, although the tumor

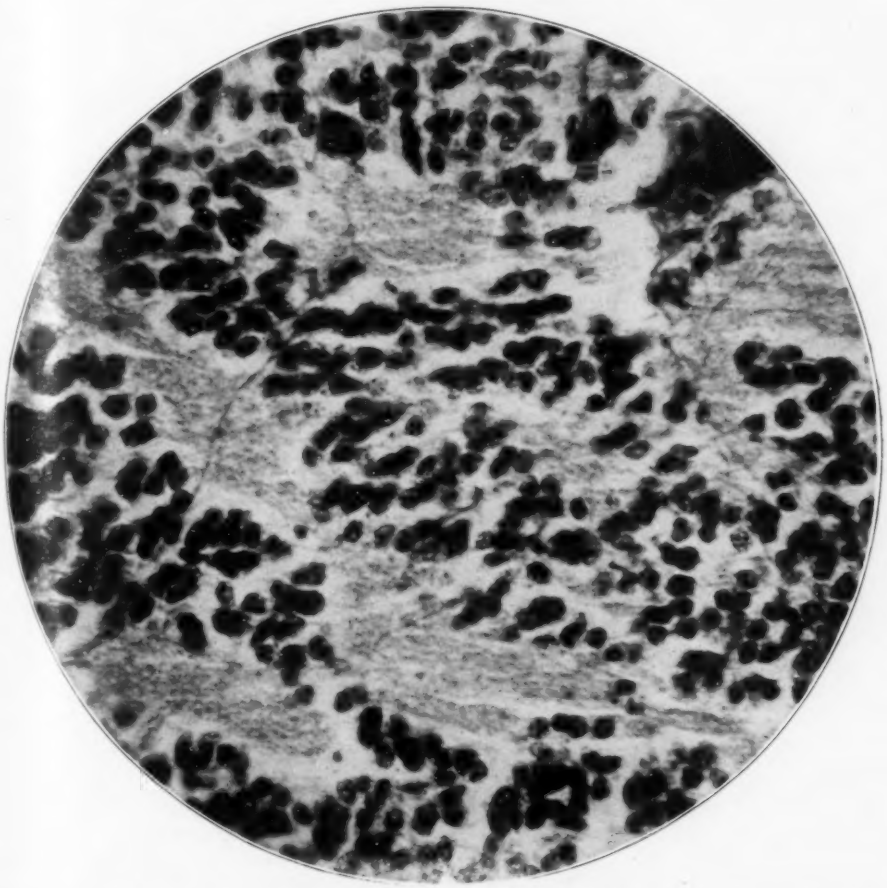

Fig. 3 (case 1).—Neuroglia fibers stained with Mallory's phosphotungstic acid hematoxylin; oil immersion.

appeared moderately vascular. One side of the section was bordered by a capsule of connective tissue. The tumor was a glioma of the pineal gland.

Case 2.—*History.*—A woman, aged 45, entered Manhattan State Hospital in June, 1909. Her father died at the age of 63 of apoplexy. A maternal aunt was insane. The patient had been normal until menstruation began, at the age of 13. Shortly after this, she began to have frequent epileptic seizures, which recurred until she was 30. During these attacks she would froth at the mouth and fall, and she would sleep for several hours afterward. Between the ages of 30 and 35, the convulsive seizures ceased, but after the latter age they again

became frequent and the patient experienced attacks of vertigo and restriction of upward rotation of the eyes. She also complained of nervousness of about eighteen years' duration.

At 39, the nervousness and dizziness increased, and the patient began to drink whisky excessively. She complained of severe pains in the occipital region and was continually moving her hands and feet. She also had several attacks of peripheral neuritis. At the age of 45, the nervousness became more pronounced, and she complained of general malaise and dizziness and would talk to imaginary people. She became completely disoriented and began to vomit frequently at the time of admission to Manhattan State Hospital, in June, 1909.

Physical Examination.—On entry, the patient was well nourished, with firm muscles and knee reflexes exaggerated and equal. There was a slight tremor of the tongue, fingers and muscles of the mouth, although writing and speech were not interfered with. The pupils were unequal, the left larger than the right, and they reacted sluggishly to light and promptly in accommodation. There was diplopia on looking downward. The patient complained of frequent headaches over the right eye. The right labial fold was slightly flatter than the left.

Ophthalmoscopic examination showed a slight haziness of the disk and surrounding retina. The tongue extended in the midline. Plantar stimulation gave flexion of the big toe. The balancing power and coordination were good, and there was no loss of power in the muscles. The ankles were rigid, and the gait was stiff. There was some tenderness of the muscles of the calf on pressure. The patient had some difficulty in distinguishing the point from the head of a pin, but could differentiate hot and cold and could localize touch. Lumbar puncture showed fluid under considerable pressure, with a marked lymphocytosis. There was no external evidence of syphilis.

Course.—At the time of entry to the hospital, there was vomiting at night and headaches with pain down the spine and in the arms. The vomiting attacks continued until October 4, when she began to tremble and lost consciousness for a few minutes. Physical examination on October 20 showed the pupils dilated, more markedly on the left, with normal reaction to light and sluggish to distance. She was unable to read large print, and hearing was poor. There was no impairment of touch and temperature sense. When placed on her feet, the patient tended to fall backward and to the right. The normal reflexes were hyperactive, but there was no response to plantar stimulation. The tongue and fingers showed slight tremor.

On November 15, the patient had a peculiar seizure in which the head was drawn to the extreme right and the eyes were fixed, but soon closed. No jerking was noted at this time.

The patient did not respond to questions. The attack lasted about twenty minutes and was followed by a return of the former condition. On December 4, the patient had an attack of twitching of the face, arms and legs, and on December 6, was found in a rigid position with the neck thrown back. A general convulsion occurred on December 23, and death took place on December 27.

Clinical Diagnosis.—The case was described as presenting the syndrome of Korsakoff in an epileptic patient of alcoholic habits and with evidence of syphilitic disorder of the nervous system.

Autopsy.—The body was that of a somewhat emaciated female adult. The pupils were circular and equal. The body cavities were normal except for pleural adhesions, which were more marked on the right. The heart was enlarged to

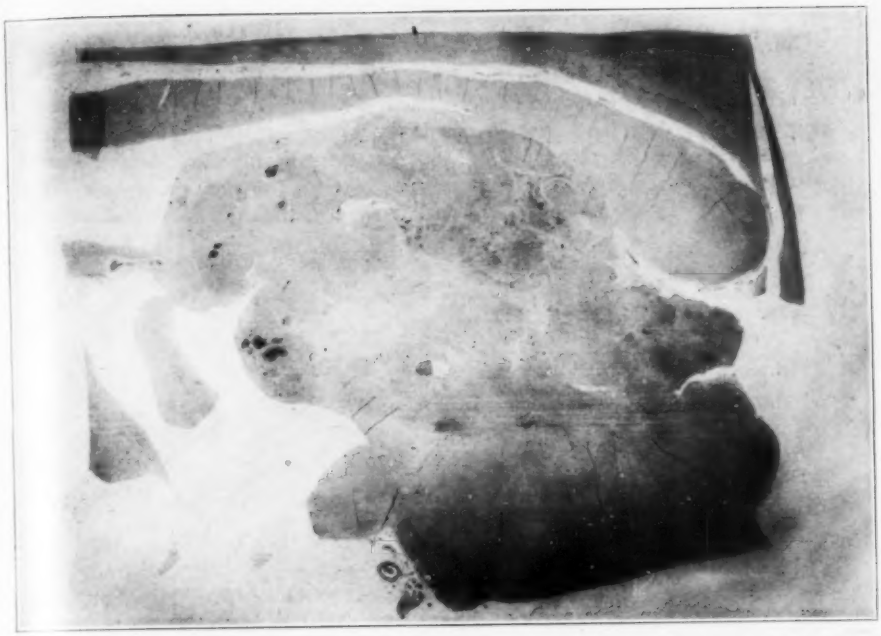

Fig. 4 (case 2).—Section taken through tumor of pineal gland which lies beneath the splenium of the corpus callosum, seen at the top of the section.

Fig. 5 (case 2).—Series of sections through the spinal cord, showing a syringomyelic cavity extending through the cervical and thoracic regions.

the right, and the mitral valve showed a slightly nodular thickening. The right lung contained numerous small abscess cavities, surrounded by areas of consolidation, while the left lung showed compensatory emphysema. The spleen was slightly enlarged and hyperemic. The liver was small and slightly opaque, and the gallbladder contained a few stones. The kidneys were normal except for a serpiginous scar on the posterior surface of the right kidney. The suprarenals and pancreas were normal.

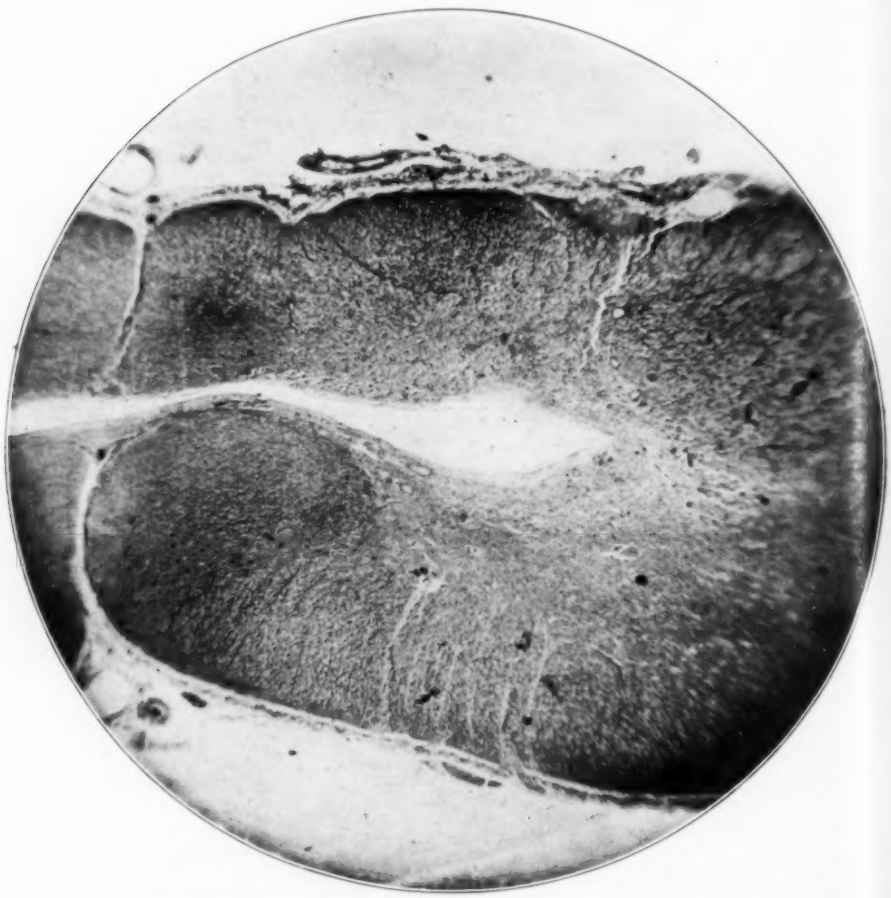

Fig. 6 (case 2).—Section through the cervical region of the spinal cord, showing the syringomyelic cavity. This is a part of the section shown at the right end of the upper row in figure 5.

Examination of the pelvis showed numerous adhesions and multiple uterine myomas. Occasional small atheromatous plaques were found in the aorta. The gastro-intestinal tract was normal.

The calvarium was symmetrical superficially and was 3 mm. thick. The under surface of the dura was normal, and the amount of cerebrospinal fluid was reduced. The brain as a whole appeared compressed outward; the convolutions were flattened, and multiple tiny hernias had broken through the dura and slightly

eroded the bone, being most pronounced in the left frontal region. The bone underlying the frontal lobes appeared somewhat thinned; the opening into the sella was enlarged and the hypophysis compressed and crescent-shaped on section, resting on extremely thin bone.

The brain, aside from the general appearance of internal pressure, seemed to be of fair size without superficial asymmetries. The tuber cinereum bulged outward and forward like a thin-walled bubble and extended into the sella. The anterior portions of the gyri recti were thin and appeared wedged on either side of the crista galli. The inferior portion of the cerebellum showed a depression

Fig. 7 (case 2).—Section through tumor, showing the border of an area of necrosis. Hematoxylin and eosin stain; low magnification.

corresponding to the wedging of the brain into the foramen magnum. The basal vessels were free from atheroma, and the fourth ventricle did not show any granulations.

Separation of the walls of the two hemispheres revealed a tumor occupying the posterior and superior two thirds of the third ventricle, completely occluding the aqueduct of Sylvius and extending forward as far as the posterior edge of the foramen of Monro and upward so as to elevate the posterior third of the corpus callosum. Parallel sagittal sections showed the growth to extend about 2.5 cm. to the right and 2 cm. to the left of the midline, and to infiltrate extensively the thalamus, particularly the pulvinar.

The anterior portion of the third ventricle and the lateral ventricles were considerably dilated. No gross changes appeared in cross sections of the optic nerves. Grossly, in the tumor one could not distinguish the pineal gland.

The spinal cord presented a distinct flattening in the cervical region, and on section a transversely running cavity was seen which became smaller in the thoracic region and disappeared on cross section of the lumbar region. The pia of the cord appeared normal grossly. A microscopic section through the cervical cord showed a few lymphoid cells in the pia, while in the lumbar region many

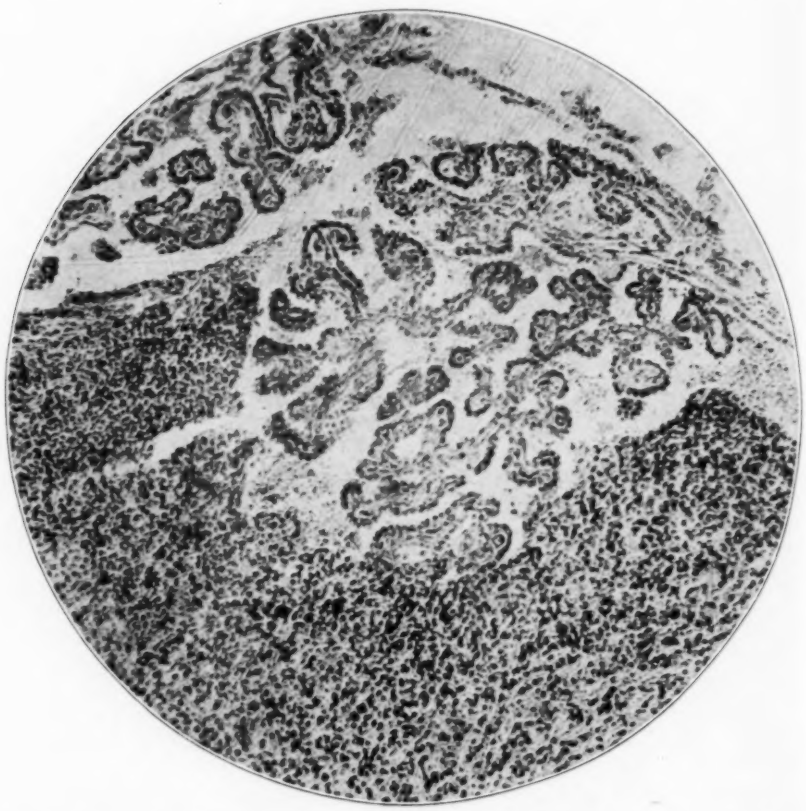

Fig. 8 (case 2).—Fragments of choroid plexus embedded in one border of the tumor. Hematoxylin and eosin stain; low magnification.

lymphocytes, some plasma cells and accumulations of endothelioid cells were found, as well as areas showing beginning necrosis. There was a marked inflammatory infiltration of the walls of the veins in the pia.

A sagittal section of the tumor was made and included also the splenium of the corpus callosum and the adjacent dorsal part of the medulla. The section of the tumor was 4 cm. anteroposteriorly, and 2 cm. in the vertical diameter. The tumor pressed against but did not invade the corpus callosum. There was no sharp line of demarcation between the tumor and the corpora quadrigemina, the appearance being that of an invasion of the latter.

Microscopically, the picture varied somewhat as to cellularity and vascularity in different parts of the tumor, but the fundamental structure was the same throughout. There was a ground substance of neuroglia fibrils through which were thickly scattered nuclei ranging in size from 7 to 14 microns. The nuclei were round or slightly oval and were uniformly stippled with chromatin, the nucleoli being inconspicuous. Around some of the nuclei, faintly staining cell bodies could be seen sending out irregular processes. The nuclei appeared for the most part singly or in pairs, with the occasional grouping together of nuclei in

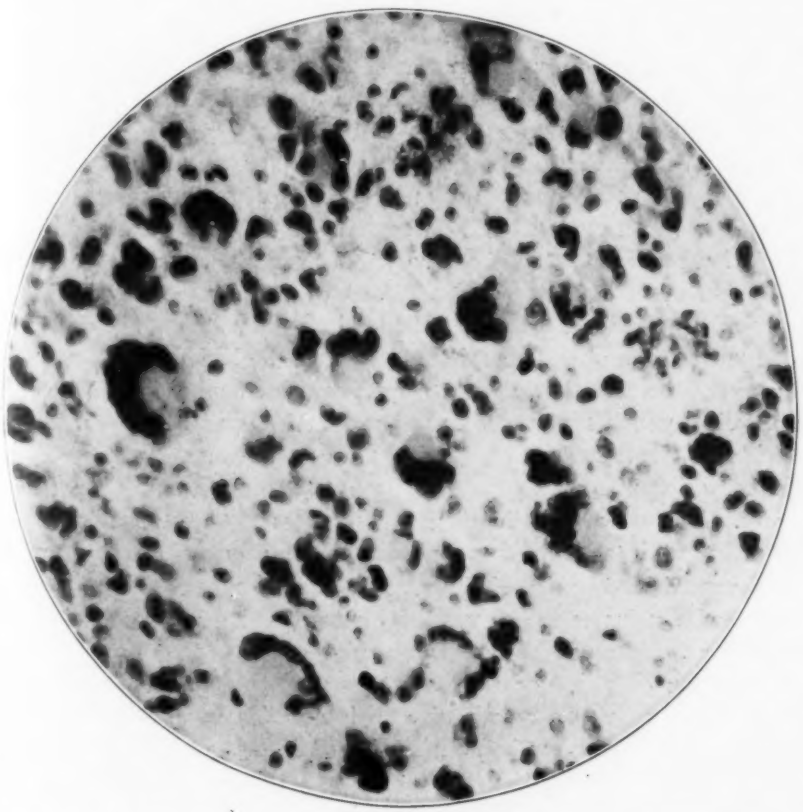

Fig. 9 (case 2).—Multinuclear giant cells. Hematoxylin and eosin stain; oil immersion.

clumps or semicircles. In certain areas, these nuclei formed numerous multinuclear giant cells with clear cytoplasm. Four or five nuclei were often arranged in a semicircle at one side of the giant cells and constituted about one half of its area. A few of the giant cells contained large vacuoles. No mitotic figures were seen.

The tumor was fairly well supplied with blood vessels, which in places showed marked proliferation. Many of the blood vessels were thrombotic, particularly those near areas of necrosis, and there were occasional small extravasations of blood. In the center of the tumor was an area of coagulative necrosis, containing and surrounded by thrombotic vessels, measuring about 4 by 10 mm.,

while smaller areas of like nature were seen in various parts of the tumor. The perivascular fibrous tissue contained large numbers of mononuclear phagocytic cells loaded with pigment, apparently hemosiderin.

Fragments of choroid plexus extended into the tumor along fissures in its substance. A few isolated bits of choroid plexus were seen surrounded by tumor tissue, although not far removed from the larger fragments. None of this choroid plexus showed evidence of malignant proliferation. A few small spherules of calcium with concentric lamination were seen in various parts of the tumor. This tumor also was a glioma of the pineal gland.

The finding of a syringomyelic cavity in case 2 is of considerable interest since, according to the "stasis hypothesis" of Langhans, such a cavity in the spinal cord may result from increased intracranial pressure, particularly that due to a tumor in the posterior cranial fossa. He reports four cases of tumor of the posterior fossa associated with syringomyelia: (1) a secondary melanosarcoma in the floor of the fourth ventricle; (2) a sarcoma of the vermis; (3) a sarcomatous degeneration of the choroid plexus of the fourth ventricle, and (4) a marked development of both cerebellar tonsils to form two pyramidal, symmetrical tumors which displaced the medulla oblongata. The production of cavities in the spinal cord in such cases is, he believes, due to forcing the cerebellum into the foramen magnum with compression of the vertebral veins, which leads to venous stasis and edema of the cord. This stasis and edema favor distention of the central canal, hydromyelia, and disintegration of the gray matter of the cord to produce a syringomyelic cavity. He believes that in some cases the syringomyelic cavity may be formed as a diverticulum from the dilated central canal.

Kronthal advanced a similar theory for the association of syringomyelia with a tumor in the vertebral canal causing venous stasis in the spinal cord. As a result, there is widening of the central canal and later proliferation of glia, due to the poorer nourishment. When the glial proliferation reaches a certain size, disintegration must take place since its center can no longer be nourished, and in this way a syringomyelic cavity is produced.

The association of cavities of the spinal cord with intracranial tumors is certainly rare; no mention of such a condition was found in any of the cases of pineal tumor reviewed, nor do recent authors on the subject of syringomyelia place importance on increased intracranial pressure as a possible etiologic factor.

In case 2, there was also found infiltration with lymphocytes, plasma cells, and endothelioid cells in the pia of the spinal cord, together with areas of beginning necrosis and inflammatory infiltration of the walls of the veins in the pia. It is evident, therefore, that the chronic meningomyelitis in this case may have a more important bearing than the pineal tumor on the cavity found in the cervical and thoracic cord.

GENERAL COMMENT

Before discussing pathologic conditions in the pineal gland, I shall give a brief review of the work that has been done on its normal structure and function. The best description of the morphology and significance of the pineal gland is to be found in the monograph by Tilney and Warren, in 1919. They conclude that: 1. The pineal region is preponderatingly glandiferous in its derivatives. 2. The pineal body is not a vestige, but is a glandular structure necessary to metabolism. 3. In cyclostomes, amphibia, and primitive reptiles, the pineal organ may be differentiated in the interest of sensory mechanism which has or had a visual function. 4. There is no direct relation between the parietal eye and the pineal body.

The secretory function is questioned by Cowdry, who points out that the pineal gland is never, at any stage in phylogeny, associated with the alimentary tract, but invariably develops from ectoderm which possesses sensory rather than internal secretory potencies. The function of the pineal gland has been a matter of much speculation since the time of Galen, who wrote, "It is in substance glandular and was devised for the same purpose as the other glands of the body." Another view, long since abandoned, was that the pineal gland acted as a valve between the third and fourth ventricles to control the flow of spinal fluid. In 1662, Descartes mentioned the pineal gland as the site of the soul.

Three methods of approach have been employed in the endeavor to determine the significance of the pineal gland: the feeding of pineal substance to animals and humans; the experimental removal or destruction of the gland in animals, and the correlation of clinical observations in cases of tumors of the pineal gland.

Results of Feeding Pineal Gland.—The first feeding experiments were those of Dana and Berkeley, who worked with young guinea-pigs, kittens and rabbits. In all the experiments, the subjects made greater gains in weight than did the controls. McCord obtained similar results with guinea-pigs, but reported that pineal feeding was without effect after maturity was reached. Hoskins was unable to affect the growth of white rats by feeding pineal gland. Sisson and Finney likewise reported negative results with rats.

Extracts of the pineal gland have been fed to defective children with indefinite, though somewhat encouraging, results. Goddard and Cornell gave dried pineal gland to a group of twenty-five mentally defective children over a period of four months. At the end of this period, these children had made a mental improvement on an average slightly above that made by a similar group of defective children who had not received any gland.

Results of Destruction of Pineal Gland in Animals.—Experimental work on the effect of extirpation of the pineal gland has been handi-

capped by the high mortality incident to that procedure. Sarteschi destroyed the pineal gland in rabbits, with negative results due to failure of the animals to survive the operation. In later work, the same author produced hypertrophy of the testes in young rabbits and puppies by removal of the pineal gland.

Foa removed the pineal glands of young chicks and found that the survivors showed excessive growth of testes and crests. He also produced hypertrophy of the testes and a general somatic overdevelopment in the rat by ablation of the pineal gland, but little effect was produced in the female. Horrax worked with guinea-pigs and rats. He reported a hastened development of the testes in the experimental animals.

Dandy perfected a method whereby the pineal gland can be reached and destroyed through the third ventricle, with decreased mortality. Following the removal of the pineal gland in the young puppies, he observed no sexual precocity or indolence, no adiposity or emaciation, no somatic or mental precocity or retardation. He concluded that "The pineal is apparently not essential to life and seems to have no influence on the animal's well being."

Symptoms of Tumor of the Pineal Gland.—The clinical observations, in cases proving at autopsy to be tumors of the pineal gland have given rise to numerous speculations regarding the functions of the gland. While the effect of such a tumor in an adult is limited to local and general pressure symptoms, the picture in preadolesent cases presents a striking contrast and has pointed to a relationship between the pineal gland and the development of the gonads, especially in males.

Whether the premature development of secondary sex characteristics is due to hypopinealism or hyperpinealism is still a matter of dispute. That the former is more probable is indicated by the fact that the pineal gland atrophies at puberty, that the syndrome of "macrogenitosomia praecox" occurs in children in whom autopsy shows replacement of the pineal gland by a tumor, and finally by the hastened sexual maturity resulting from experimental extirpation of this gland. On the other hand, Zandren has reported a case of infantilism in a boy, aged 17, with total absence of the pineal body, from which he concludes that the function of the pineal gland is to initiate the process of puberty by the secretion of a hormone stimulating the sexual glands.

The symptoms that have been observed in cases of tumor of the pineal gland have been best described by Bailey and Jelliffe, Bell, Horrax, Jelliffe, and Robbins. These symptoms may be grouped according to the mode of production as:

1. The general symptoms of intracranial pressure: headache (especially occipital), vomiting, choked disk, mental apathy, somnolence, clonic spasms, and incontinence of urine and feces.

2. Localizing symptoms: ocular palsies, rigid dilated pupils, nystagmus, blindness (possibly due to internal hydrocephalus with pressure on the chiasma), deafness, cerebellar symptoms (ataxia, adiadokokinesis), peduncular symptoms (monoplegia, oculomotor paralysis), pontobulbar symptoms (nystagmus, disturbances of speech and swallowing), and disturbances of hypothalamic sympathetic nerve centers (exophthalmos, tachycardia, vasomotor cutaneous changes, diabetes insipidus and polyphagia).

3. Symptoms referable to the pineal gland: It has been found (table 3) that many cases of tumor of the pineal gland in young patients are characterized by a precocious development of the secondary sex characteristics. This syndrome has been designated by Pellizzi as "macrogenitosomia praecox" and described as follows: (1) precocious development and differentiation of the external genitalia with the premature appearance of axillary and pubic hair; (2) precocious development of sexual function; (3) precocious, abnormal growth of the long bones; (4) the appearance of signs of internal hydrocephalus, including visual disorders, headache, vomiting, with choked disk or optic atrophy; (5) the absence of all other motor and sensory symptoms. Bailey and Jelliffe, following Marburg, have listed as "metabolic symptoms" the adiposity, early sexual maturity and cachexia sometimes found in tumors of the pineal gland.

Surgical Removal of Tumors of the Pineal Gland.—The surgical removal of tumors in the region of the pineal gland, while difficult of accomplishment and in most cases resulting fatally, has been advocated by Dandy. He performed this operation on three patients. In the first case he found a cerebellar tumor infiltrating to such an extent that no attempt was made to remove it. In the second case an encapsulated tubercle of the pineal gland was removed, and the patient recovered from the operation but died eight months later, probably from other tubercles in the brain. In the third case the tumor of the pineal gland was large and could not be dissected away from the great vein of Galen and the two smaller veins of Galen, so these were removed with the tumor. The patient died after forty-eight hours, presumably of shock due to the magnitude of the operation.

SUMMARY OF PREVIOUSLY REPORTED CASES OF PINEAL TUMORS

The clinical and pathologic pictures obtained from reading reports of a large number of cases of pineal tumor are indefinite. It is only by careful analysis and tabulation of the more important observations that one can draw conclusions of value in the localization of such tumors. Such tables are here presented and embody all cases in the available literature.

TABLE 1.—*General Information Concerning Pineal Tumors*

Case No.	Author	Bibliography Year	Bibliography No.	Sex*	Age, Years	Type of Tumor
1	Blane	1800	13	♂	33	Fibrous tumor
2	Schmidt	1837	129	♀	22	Tumor
3	Stanley	1837	137	♀	4	Cyst
4	Schultze	1848	131	♂	69	Purulent pineal
5	Simon	1859	135	♀	35	Hemorrhage in pineal
6	Friedreich	1865	40	♂	59	Psammoma
7	Blanquinque	1871	14	♂	39	Psammoma
8	Massot	1872	96	♂	19	Carcinoma
9	Bouchut	1872	17	♂♀	2	Cyst
10	Hirtz	1875	59	♂	21	Lipoma on corpora quadrigemina
11	Schearer	1875	127	♀	16	Tumor
12	Weigert	1875	151	♂	14	Teratoma
13	Duffin and Ferrier	1876	33	♂	25	Glioma
14	Nieden	1879	110	♂♀	35	Hydrops cysticus
15	Falkson	1879	34	♂	16	Teratoma
16	Nothnagel	1879	111	♂	21	Sarcoma
17	Gowers	1879	48	♂	21	Sarcoma
18	Henrot	1883	56	♂	26	Sarcoma of pineal and pituitary
19	Turner	1885	145	..	..	Sarcoma (spindle cell)
20	Feilchenfeld	1885	35	♂	18	Sarcoma
21	Pontoppidan	1885	120	♂	31	Sarcoma
22	Schulz	1886	132	♂	28	Glioma
23	Reinhold	1886	123	♂	19	Gliosarcoma
24	Coats	1887	25	♂	13	Teratoma
25	Daly	1887	27	♂	23	Alveolar carcinoma
26	Nothnagel	1888	112	♂	17	Glioma
27	Gauderer	1889	43	♂	12	Cystic teratoma
28	Kny	1889	78	♂	32	Sarcoma (round-cell)
29	Zenner	1892	157	♂	13	Gliosarcoma
30	Schmid	1893	128	♂	21	Gelatinous tumor
31	König	1894	79	♂	35	Psammosarcoma
32	Gutzeit	1896	49	♂	8	Teratoma
33	Hoesslin	1896	61	♂	9	Sarcoma (spindle cell)
34	Lord	1898	89	♂	..	Syphilitic enlargement
35	Campbell	1899	23	♀	33	Cyst
36	Campbell	1899	23	♀	..	Cyst
37	Russell	1899	126	♀	23	Cyst
38	Russell	1899	126	..	..	Cyst
39	Garrod	1899	42	♂	16	Cyst
40	Lawrence	1899	84	♂	16	Glioma and tuberculous meningitis
41	Ogle	1899	115	♂	32	Melanotic sarcoma
42	Ogle	1899	115	♂	6	Teratoma
43	Oestreich and Slawyk	1899	116	♂	4½	Teratoma
44	Neumann, P.	1900	107	♂	27	Teratoma
45	Hempel	1901	55	♂	24	Alveolar carcinoma
46	Neumann, M.	1901	108	♂	28	Hydrops cysticus
47	Neumann, M.	1901	108	♂	11	Cystic sarcoma
48	Joukovsky	1901	74	♀	6 days	Cyst
49	Holzhauer	1903	62	♂	4	Sarcoma
50	Meyer	1905	100	♂	24	Adenoma
51	Askanazy	1905	2	♂	19	Chorio-epithelioma
52	Verger	1907	147	♂	42	Gliosarcoma
53	Chirone	1907	24	..	..	Hydrops cysticus
54	Marburg	1907	94	♀	9	Mixed tumor (glia and ependyma)
55	Hart	1909	50	♂	24	Angiosarcoma
56	Frankl-Hochwart	1909	39	♂	5½	Teratoma
57	Pappenheimer	1910	117	♂	10	Ependymal neuroglioma
58	Raymond and Claude	1910	122	♂	10	Glioma
59	Howell	1910	67	♂	32	Gliosarcoma
60	Howell	1910	67	♂	22	Gliosarcoma
61	Howell	1910	67	♂	20	Gliosarcoma
62	Apert and Porak	1911	1	♀	37	Tumor
63	Bailey and Jelliffe	1911	5	♂	12	Teratoma
64	Dana and Berkeley	1913	28	♂	40	Fibrosarcoma
65	Goldzieher	1913	45	♂	16	Angiosarcoma
66	Hedenius and Henschen	1913	51	♂	31	Glioma
67	Hijmans Van Den Bergh and Van Hasselt	1913	58	♂	8	Teratoma
68	Hueter	1913	68	♂	19	Teratoma
69	Rohrschach	1913	125	♂	27	Mixed tumor
70	Takeya	1913	140	♂	10	Teratoma
71	Fukuo	1914	41	♂	19	Teratoma
72	Pussep	1914	121	..	..	Cyst
73	Schmincke	1914	130	♂	19	Teratoma
74	Schmincke	1914	130	♂	22	Teratoma
75	Odermatt	1915	114	♂	10	Teratoma
76	Bell	1916	8	♂	33	Hyperplasia with cysts
77	Bell	1916	8	..	..	Hyperplasia
78	Horrax	1916	64	♂	12	Hyperplasia
79	Dercum	1916	31	..	..	Tumor

* In this table and in tables 4 and 7, ♂ indicates male; ♀, female

TABLE 1.—*General Information Concerning Pineal Tumors—Continued*

Case No.	Author	Bibliography Year	Bibliography No.	Sex	Age, Years	Type of Tumor
80	Uemura	1917	146	♂	50	Fibrosarcoma
81	Uemura	1917	146	♀	30	Glioma
82	Uemura	1917	146	..	..	Adenoma
83	Skoog	1918	136	♂	9	Hyperplasia
84	Boehm	1919	16	♂	9	Teratoma
85	Berblinger	1920	9	♂	35	Glioma
86	Frank	1920	37	♂	20	Teratoma
87	Löwenthal	1920	90	♂	27	Hyperplasia
88	Businco	1921	22	♀	28	Cystic neuroglioma
89	Dandy	1921	29	..	..	Tubercle in pineal
90	Dandy	1921	29	..	..	Large tumor
91	Giebel	1921	44	..	..	Sarcoma
92	Giebel	1921	44	..	..	Dermoid tumor
93	Jacobi	1921	69	♀	21	Metastatic sarcoma from ovary
94	Klapproth	1921	76	♂	15	Teratoma
95	Lereboullet, Maillot and Brizard	1921	88	♂	12	Neuro-epithelioglioma
96	Luce	1921	91	♀	9	Teratoma
97	Luce	1921	91	♀	26	Angiosarcoma
98	Tilney and Riley	1921	141	♂	8	Tumor
99	Steiner and Johan	1922	138	♂	7	Adenoma
100	Wilson	1922	155	♂	23	Carcinoma of pineal arising from choroid plexus
101	De Monchy	1923	101	♂	14	Teratoid tumor
102	Brusa	1924	21	♂	3	Tumor
103	Horrax and Bailey	1925	65	♂	28	Pinealoma (spongioblastic)
104	Horrax and Bailey	1925	65	♀	29	Pinealoma (spongioblastic)
105	Horrax and Bailey	1925	65	♂	6	Pinealoma (spongioblastic)
106	Horrax and Bailey	1925	65	♀	10	Pinealoma (spongioblastic)
107	Horrax and Bailey	1925	65	♂	3	Teratoma
108	Horrax and Bailey	1925	65	♂	23	Pinealoma (adult type)
109	Horrax and Bailey	1925	65	♂	13	Pinealoma (adult type)
110	Horrax and Bailey	1925	65	♂	17	Pinealoma associated with glioma of third ventricle
111	Horrax and Bailey	1925	65	♂	20	Pinealoma (adult type)
112	Horrax and Bailey	1925	65	♀	15	Pinealoma (spongioblastic)
113	Horrax and Bailey	1925	65	♂	24	Pinealoma (adult type)

TABLE 2.—*Symptomatology*

Case No.	Sexual Precocity	Blindness or Impaired Vision	Ocular Palsies	Other Symptoms
1	.....	.....	.....	Headache, delirium
2	.....	.....	Dilated sluggish pupils.....	Headache, vertigo
3	.....	.....	Left pupil fixed, contracted; right pupil irregularly dilated	Vomiting, coma
4	.....	Blindness	.....	Headache
5	.....	Blindness	.....	Headache
6	.....	.....	.....	Headache
7	.....	Blindness	Conjugate deviation below and to right	Headache, epileptiform crises
8	.....	Impaired vision	Diplopia, internal strabismus, paralysis of upward movements	Headache, convulsions, vomiting, polyuria
9	.....	.....	Conjugate deviation, pupils sluggish to light	Convulsions, coma
10	.....	Blindness	.....	Headache, vomiting, coma
11	.....	.....	.....	Epilepsy
12	.....	.....	.....	.....
13	.....	Impaired vision	Right internal squint, diplopia, sluggish pupils	Headache, vertigo, coma
14	.....	Impaired vision	Diplopia, paralysis of right fourth nerve	Vertigo
15	.....	Impaired vision	Paralysis of both superior recti and left internal rectus, diplopia	Drowsiness
16	.....	Blindness	Pupils dilated, eyes immobile....	Epilepsy, coma
17	.....	.....	Paralysis of sixth cranial nerve	Headache, deafness, facial paralysis
18	.....	.....	.....	.....
19	.....	.....	.....	.....
20	.....	.....	Diplopia, paralysis of all ocular muscles	Headache, convulsions, vomiting, coma
21	.....	.....	Conjugate deviation to right, limited eye movements	Headache, vertigo
22	.....	Impaired vision	.....	Headache, staggering gait

TABLE 2.—*Symptomatology—Continued*

Case No.	Sexual Precocity	Blindness or Impaired Vision	Ocular Palsies	Other Symptoms
23	.....	Impaired vision	Bilateral ptosis, paralysis of sixth nerve	Headache, vertigo, nystagmus
24	.....	.....	Ptosis of left lid, paresis of right internal rectus	Epileptiform attacks, drowsiness, vomiting
25	.....	Impaired vision	Sluggish pupils, paralysis of left internal rectus	Headache, convulsions, vomiting
26	.....	Blindness	Sluggish pupils	Headache, vertigo, nystagmus
27	.....	.....	Sluggish reaction to light.....	Headache, vomiting, stupor, opisthotonos
28	.....	Blindness	Rigid pupils, divergent strabismus	Headache, convulsions, lateral nystagmus
29	.....	Blindness	Pupils dilated and immobile to light	Headache, stupor, deaf and dumb
30	.....	.....	.....	.....
31	.....	.....	.....	.....
32	Present	.....	Rigid pupils, diplopia, paresis of left internal rectus, paralysis of downward movements	Headache, vomiting, deafness, nystagmus
33	.....	Blindness	Paralysis of both superior recti, and superior oblique, rigid puplis	Headache, convulsions, vomiting, thirst, polyuria
34	.....	.....	.....	Epilepsy, hemiplegia, stupor
35	.....	.....	.....	Epilepsy
36	.....	.....	.....	Epilepsy
37	.....	.....	.....	Fracture of base of skull
38	.....	.....	.....	.....
39	.....	.....	.....	.....
40	.....	.....	Pupils dilated and rigid.....	Headache, delirium, nystagmus
41	.....	.....	.....	.....
42	Present	Blindness	Pupils dilated and rigid, paresis of right external rectus	Vomiting, somnolence
43	Present	.....	Right convergent strabismus, sluggish reaction to light and in accommodation	Stupor, convulsions
44	.....	Impaired vision	Diplopia, paralysis of movements to right and upward, rigid pupils	Headache, vomiting, deafness, nystagmus
45	.....	.....	Argyll Robertson pupils.....	Headache, stupor, contractures, rotary nystagmus
46	.....	Impaired vision	Rigid dilated pupils, ptosis of left lid, internal convergence	Vomiting, deafness
47	.....	.....	.....	Headache, vomiting, fever
48	.....	.....	Ptosis, divergent strabismus....	Continuous sleep
49	Present	.....	.....	.....
50	.....	Blindness	.....	Convulsions, flow of cerebrospinal fluid from nose, left-sided facial paralysis
51	.....	.....	Right pupil dilated, left contracted	Headache, vertigo, vomiting
52	.....	.....	.....	Headache, dementia
53	.....	.....	.....	.....
54	.....	Impaired vision	.....	Headache, vomiting, adiposity
55	.....	Impaired vision	.....	Headache, nausea
56	Present	Impaired vision	Sluggish pupils, strabismus, bilateral paralysis of superior, inferior and lateral recti	Headache, stupor, vomiting, right facial paralysis
57	.....	Impaired vision	.....	Headache, vomiting
58	.....	Blindness	Pupils dilated and rigid.....	Headache, adiposity
59	.....	Impaired vision	Diplopia, paralysis of upward movement, small rigid pupils	Headache
60	.....	.....	Diplopia, paralysis of upward movement, rigid pupils	Nausea, thirst, stupor
61	.....	.....	Paralysis of upward movement	Headache, failing memory, nystagmus
62	.....	Blindness	.....	Headache, vomiting
63	.....	Impaired vision	Paralysis of upward movement, sluggish pupils	Stupor, adiposity, physical overgrowth, nystagmus
64	.....	Impaired vision	.....	Headache, vertigo, falling
65	Present	.....	.....	.....
66	.....	.....	Convergent strabismus, diplopia	Headache, positive Romberg sign
67	Present	.....	.....	.....
68	.....	Impaired vision	.....	Headache, vertigo, vomiting, convulsions
69	.....	.....	.....	Character changes

TABLE 2.—*Symptomatology—Continued*

Case No.	Sexual Precocity	Blindness or Impaired Vision	Ocular Palsies	Other Symptoms
70	Present	.....	.....	.....
71	.....	.....	.....	.....
72	.....	.....	.....	.....
73	.....	.....	.....	.....
74	.....	.....	.....	General symptoms of intracranial pressure
75	Present	.....	.....	General symptoms of increased intracranial pressure
76	.....	.....	.....	Cardiac decompensation
77	.....	.....	.....	.....
78	Present	.....	Diplopia, unequal pupils.....	Headache, vomiting, staggering to right, right-sided numbness, convulsions, polyuria, nuchal rigidity
79	.....	.....	.....	.....
80	.....	.....	.....	.....
81	.....	.....	.....	Epilepsy
82	.....	.....	.....	.....
83	Present	.....	Diplopia	Headaches
84	Present	.....	Paresis of both sixth nerves, inequality of pupils	General pressure symptoms
85	.....	.....	Paralysis of third and fourth nerves	Headache, facial paralysis
86	.....	.....	Rigid pupils	Polydipsia, rhythmic contraction of soft palate
87	.....	.....	.....	Adiposity
88	.....	.....	.....	.....
89	.....	.....	.....	.....
90	.....	.....	.....	.....
91	.....	.....	.....	.....
92	.....	.....	.....	.....
93	.....	.....	Rigid pupils, ptosis of left eye, paresis of right internal rectus muscle and left sixth nerve	Headache, left facial paralysis
94	.....	.....	.....	Headache, infantile habitus, small scrotum
95	Present	Impaired vision	Limited upward movement.....	Headache, vomiting, somnolence
96	.....	.....	.....	.....
97	.....	.....	.....	.....
98	Present	Impaired vision	.....	Headache, vomiting, convulsions
99	Present	.....	Bilateral sixth nerve paralysis	Vertigo, vomiting
100	.....	.....	Diplopia, Argyll Robertson pupils, paralysis of upward and downward movement	.....
101	.....	.....	Rhythmic convergence spasm, Argyll Robertson pupils, paralysis of upward and downward movement	Adiposity, small genitalia
102	.....	.....	.....	General symptoms of high intracranial pressure
103	.....	Blindness	.....	Tendency to fall backward, deafness
104	.....	Blindness	Diplopia	Deafness, convulsions, right facial paresis
105	.....	Impaired vision	Left abducens palsy.....	Headaches, vomiting, cervical rigidity
106	.....	Impaired vision	Rigid dilated pupils, bilateral abducens paralysis	Headaches, vomiting, staggering, deafness
107	Physical and mental precocity	Impaired vision	Paralysis of conjugate upward movement, right abducens palsy	Headaches, vomiting, convulsions, nystagmus
108	.....	Impaired vision	Bilateral abducens paresis.....	Headaches, bilateral deafness, numbness of right arm, leg and face, nystagmus, ataxia
109	.....	Impaired vision	Paralysis of upward movement	Headaches, vomiting, polydipsia, polyuria
110	.....	.....	Diplopia, rigid pupils, bilateral ptosis	Polydipsia, polyuria, headaches, vomiting
111	.....	Blindness	Paralysis of upward movement, bilateral abducens paralysis	Headaches, polydipsia, polyuria, vomiting, nystagmus, slight deafness
112	.....	Blindness	Diplopia, paresis of right internal rectus, paralysis of upward movement	Headaches, hypesthesia of right side of face
113	.....	.....	Left abducens paralysis.....	Headaches, vomiting, dizziness, left facial weakness, cervical rigidity, dysarthria

TABLE 3.—*Tabulation Based on Microscopic Pictures*

A. Teratomas of the Pineal Gland—22 Cases

Author	Year	Author	Year
Weigert	1875	Hueter	1913
Falkson	1879	Takeya	1913
Coats	1887	Fukuo	1914
Gauderer	1889	Schmincke	1914
Gutzeit	1895	Schmincke	1914
Ogle	1899	Odermatt	1915
Oestreich and Slawyk	1899	Boehm	1919
Neumann	1900	Frank	1920
Frankl-Hochwart	1909	Klapproth	1921
Bailey and Jelliffe	1911	Luce	1921
Hijmans	1913	Horrax and Bailey	1925

B. Sarcomas of the Pineal Gland—24 Cases

Nothnagel	1879	Verger	1901
Gowers	1879	Holzhauer	1903
Henrot	1883	Hart	1909
Turner	1885	Howell	1910
Feilchenfeld	1885	Howell	1910
Pontoppidan	1885	Howell	1910
Kny	1889	Dana and Berkeley	1913
Zenner	1892	Goldzieher	1913
König	1894	Uemura	1917
Hoesslin	1896	Glebel	1921
Ogle	1899	Jacobi (metastatic)	1921
Neumann	1899	Luce	1921

C. Cysts of the Pineal Gland—14 Cases

Stanley	1837	Russell	1899
Bouchut	1872	Garrod	1899
Nieden	1879	Neumann	1901
Reinhold	1886	Joukovsky	1901
Campbell	1899	Chirone	1907
Campbell	1899	Pussep	1914
Russell	1899	Bell	1916

D. Gliomas of the Pineal Gland—11 Cases

Duffin	1876	Hedenius and Henschen	1913
Schulz	1886	Uemura	1917
Nothnagel	1888	Berblinger	1920
Lawrence	1899	Businco	1921
Pappenheimer	1910	Lereboullet	1921
Raymond and Claude	1910		

E. Pinealoma (Spongioblastic Type)—6 Cases

Horrax and Bailey	1925	Horrax and Bailey	1925
Horrax and Bailey	1925	Horrax and Bailey	1925
Horrax and Bailey	1925	Horrax and Bailey	1925

F. Pinealoma (Adult Type)—4 Cases

Horrax and Bailey	1925	Horrax and Bailey	1925
Horrax and Bailey	1925	Horrax and Bailey	1925

G. Hyperplasia of the Pineal Gland—4 Cases

Bell	1916	Skoog	1918
Horrax	1916	Löwenthal	1920

H. Carcinomas of the Pineal Gland—4 Cases

Massot	1872	Hempel	1901
Daly	1887	Wilson	1922

I. Adenomas of the Pineal Gland—4 Cases

Meyer	1905	Steiner and Johan	1922
Uemura	1917	De Monchy	1923

J. Psammomas of the Pineal Gland—2 Cases

Friedreich	1865	Blanquinque	1871

TABLE 3.—*Tabulation Based on Microscopic Pictures—Continued*

K. Unclassified Tumors of the Pineal Gland—18 Cases

Author	Year	Description of Tumor
Blane	1800	Fibrous tumor
Schmidt	1837	
Schultze	1848	Purulent pineal
Simon	1859	Hemorrhage
Hirtz	1875	Lipoma
Schearer	1875	
Schmid	1893	Gelatinous tumor
Lord	1898	Syphilitic enlargement
Askanazy	1906	Chorio-epithelioma
Marburg	1907	Mixed tumor
Apert and Porak	1911	
Rorschach	1913	Mixed tumor
Dercum	1916	
Dandy	1921	Encapsulated tubercle of pineal
Dandy	1921	
Giebel	1921	Dermoid tumor
Brusa	1924	

TABLE 4.—*The Sixteen Cases Showing Macrogenitosomia Praecox*

Case No.	Author	Year	Sex	Age, Years	Type of Tumor
32	Gutzeit	1896	♂	8	Teratoma
42	Ogle	1899	♂	6	Teratoma
43	Oestreich and Slawyk	1899	♂	4½	Teratoma
49	Holzhauer	1903	♂	4	Sarcoma
56	Frankl-Hochwart	1909	♂	5½	Teratoma
65	Goldzieher	1913	♂	16	Angiosarcoma
67	Hijmans Van Den Bergh and Van Hasselt	1913	♂	8	Teratoma
70	Takeya	1913	♂	10	Teratoma
75	Odermatt	1915	♂	10	Teratoma
78	Horrax	1916	♂	12	Hyperplasia
83	Skoog	1918	♂	9	Hyperplasia
84	Boehm	1919	♂	9	Teratoma
95	Lereboullet	1921	♂	12	Neuro-epithelioglioma
98	Tilney and Riley	1921	♂	8	Unclassified
99	Steiner and Johan	1922	♂	7	Adenoma
107	Horrax and Bailey	1925	♂	3	Teratoma

TABLE 5.—*Summary of Eye Signs and Symptoms*

	Cases		Cases
Blindness or impairment of vision	45	Conjugate deviation	3
Diplopia	16	Paralysis of fourth cranial nerve	3
Paralysis of upward movement	15	Paralysis of all extra-ocular muscles	3
Paralysis of sixth cranial nerve	14	Paralysis of downward movement	3
Immobility of one or both pupils	14	Unilateral ptosis	3
Nystagmus	12	Bilateral ptosis	3
Internal strabismus	5	External strabismus	1
Paralysis of third cranial nerve	4	Constriction of pupil	1
Inequality of pupils	4	Rhythmic convergence spasm	1
Argyll Robertson pupils	3		

TABLE 6.—*Incidence of Tumors According to Age and Sex*

Age, Years	Total Cases	Male	Female
From 0 to 5	8	5	3
6 to 10	16	13	3
11 to 15	12	11	1
16 to 20	17	16	1
21 to 30	28	20	8
31 to 40	14	9	5
41 to 50	2	1	1
Over 50	3	3	0
Age not given	2	0	2
Age and sex not given	11		
Total	113	78	24

TABLE 7.—*Absence or Hypoplasia of Pineal Gland*

Author	Bibliography Year	Bibliography No.	Sex	Age, Years	Pathology	Symptoms
Askanazy and Brack	1921	4	♀	21	Hypoplasia of pineal, one fourth normal weight (0.04 Gm.)	Idiocy, precocious development of mammary glands and hair, convulsions
Zandren.....	1921	153	♂	17	Total absence of pineal	Sexual infantilism

DIAGNOSIS OF TUMOR OF THE PINEAL GLAND

From a review of the reported cases of tumor of the pineal gland the question naturally arises, Can these tumors be localized antemortem? Several authors have attempted to summarize the symptoms that point to this condition. Discussing the diagnosis of pineal tumors in adults, Horrax remarks that, "In the presence of an intracranial pressure syndrome which simulates a cerebellar lesion, if there be added to the picture an involvement of one or both trochlear nerves, together with recurring changes in the size of one or both pupils, and a tendency to spasticity in the lower extremities, a tumor of the pineal body should at least be suspected."

In preadolescent children, a pineal tumor often produces the syndrome characterized by Pellizzi as "macrogenitosomia praecox" and has been described in the foregoing. In discussing the diagnosis of a tumor in young patients, Frankl-Hochwart says, "When one finds in a very young individual, along with the general symptoms of tumor, as well as the local signs of a lesion of the corpora quadrigemina, abnormal body growth, unusual growth of hair, adiposity, somnolence, premature genital and sexual development, and finally intellectual maturity, one must think of pineal tumor."

Notwithstanding the clear-cut clinical picture described for a tumor of the pineal gland in a preadolescent child, only a few of these tumors have been correctly localized antemortem. Skoog, Boehm, and Lereboullet and Brizard each report a case diagnosed as tumor of the pineal gland, because of the precocious sexual development and other localizing symptoms verified at autopsy. Numerous cases have been reported as tumors of the pineal gland in which no verification had been made at autopsy (cases of Morse, Dercum, Horrax, Warren and Tilney, Klippel, Weil and Minvielle, Hekman, and Lereboullet).

From an analysis of the signs and symptoms (table 5) in all available reports of pineal tumors, it is evident that the eye signs are an important aid in the localization of such tumors. Probably the most significant of the eye signs are: paralysis of upward movement, diplopia, abducens

paralysis, ptosis, and absence of the light reflex. The latter sign, when unaccompanied by blindness, is probably due to encroachment of the pineal tumor on the superior colliculus through which the pupillary light reflex arc passes. The other ocular palsies are important, although it is difficult to determine to what extent they are due to local pressure of the tumor, to general intracranial pressure, and to internal hydrocephalus from obstruction of the aqueduct of Sylvius.

SUMMARY

1. Two cases of glioma of the pineal gland are reported with complete clinical and pathologic characteristics.

2. In view of the contradictory results obtained in the experimental work on pineal gland feeding of animals and of defective children and on the destruction of the pineal gland in animals, it is impossible to draw any conclusions regarding the function and significance of this gland.

3. A tumor of the pineal gland in a child is occasionally accompanied by precocious sexual development and adiposity or general overgrowth, which with symptoms of internal hydrocephalus make up the syndrome designated by Pellizzi as "macrogenitosomia praecox."

4. One hundred and thirteen cases of tumor of the pineal gland are summarized in chronological order (tables 1 and 2).

5. Tabulation (table 3) of the microscopic picture presented in the 113 cases shows: sarcoma, 24 cases; teratoma, 22 cases; cyst, 14 cases; glioma, 11 cases; pinealoma, 10 cases; hyperplasia, 4 cases; carcinoma, 4 cases; adenoma, 4 cases; psammoma, 2 cases, and unclassified, 18 cases.

6. The syndrome of "macrogenitosomia praecox" was observed in sixteen cases of pineal tumor, all in males between the ages of 3 and 16 years (table 4).

7. Blindness or impairment of vision occurred in forty-five cases, or in more than one third of the total (table 2).

8. The most important of the eye signs in cases of pineal tumor are paralysis of upward movement, diplopia, abducens paralysis, nystagmus, ptosis, and absence of the pupillary light reflex (table 5).

9. Of the 102 cases in which the sex was reported, seventy-eight occurred in males and twenty-four in females (table 6).

10. The incidence was greatest during the second decade (twenty-nine cases), although twenty-eight patients were reported between 21 and 30 years of age, and twenty-four under 11 years (table 6).

11. No conclusions can be drawn from the two cases of hypoplasia or absence of the pineal gland (table 7).

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HEMANGIOMA OF THE SPINAL CORD

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Practically all who have reported cases of hemangioma of the spinal cord have referred to the lesion as most uncommon. Cobb,[1] in 1915, was perhaps the first to make a serious attempt to review the subject. He reported one case from Cushing's clinic and found reports of seven others in the literature. Since then, other investigators have given attention to the subject, and it is now possible to collect reports of twenty-one cases. Most observers have contributed one case each, but Elsberg [2] mentions six, Adson [3] three and Dandy [4] two, and Sargent [5] gives a report of four patients personally observed and operated on. I can add one typical and one atypical case.

While there are points of similarity in the clinical course in these cases, it is doubtful if anything like a typical picture can be drawn. Sargent points out that "variability in the symptoms" is a prominent feature. All cases have shown a relatively long period when motor or sensory symptoms have been slowly progressive. The climax is usually sudden, resulting in more or less complete paraplegia with sensory disturbances indicating the level of the lesion. In some instances remarkable remissions have been seen—eight years in Frazier's [6] case, and four years in Gaupp's [7] and in one of Sargent's cases. In the majority of instances, however, the remission has not been of long standing, nor has the relief of symptoms led to anything like complete return of function. In view of the sudden development of final paralysis, a diagnosis of myelitis has perhaps been most common. Tumor of the spinal cord, however, must always be given serious consideration.

That the pathologic condition has impressed various observers differently is seen when one compares the sundry terms of description

1. Cobb, Stanley: Hemangioma of the Spinal Cord, Ann. Surg. **62**:641, 1915.

2. Elsberg, C. A.: Diagnosis and Treatment of Surgical Diseases of the Spinal Cord and its Membranes, Philadelphia, W. B. Saunders Co., 1916.

3. Adson, A. W., and Ott, W. O.: Results of the Removal of Tumors of the Spinal Cord, Arch. Neurol. & Psychiat. **8**:520 (Nov.) 1922.

4. Dandy, W. E.: The Diagnosis and Localization of Spinal Cord Tumors, Ann. Surg. **81**:223, 1925.

5. Sargent, Percy: Haemangioma of the Pia Mater Causing Compression Paraplegia, Brain **48**:259, 1925.

6. Spiller, W. G., and Frazier, C. H.: Telangiectasis of the Spinal Cord, Arch. Neurol. & Psychiat. **10**:29 (July) 1923.

7. Gaupp: Zwei Neurofibrome und ein Angiom der Cauda equina, centrales Glioma and Syringomyelie des Lendenteils des Rückenmarks, Ziegler's Beitr. z. Path. **2**:510, 1888; Hemorrhoiden der Pia-mater spinalis in Gebiete des Lendenmarks, ibid. **2**:516, 1888.

employed. Hemangioma is perhaps most commonly used and as more cases come to light may gain further acceptance. The earlier observers referred to the lesion as an "angioma," a "cavernous angioma," "a mass of convoluted or tortuous veins" on the dorsal surface of the cord; "a varix or varicosity of the venous plexuses" surrounding the cord. Frazier and Spiller [6] refer to their case as one of "telangiectasis," while Sargent mentions the "nevoid" character of the vessels, which are usually venous in origin although they may occasionally be arterial. The latter type he refers to as "arterial hemangioma," "aneurysmal varix," "plexiform angioma" or "cirsoid aneurysm." His report of arterial angioma is the only one that we have found; this angioma was at the level of the seventh cervical vertebra and was associated with enlargement of some of the vessels in the neck, the transversus colli being particularly tortuous. He refers to a case of Raymond and Cestan [8] which was similar in nature and in which the arteries as well as the veins "shared in the dilatation." In this instance the mass was unusually high up, beginning near the bulbopontile junction and disappearing toward the second thoracic segment of the cord.

The majority of hemangiomas have been found on the dorsal aspect of the cord. It is supposed that they originate from the pial veins. They may be seen in the cervical region, although the low dorsal and lumbar regions are the locations of choice. The vessels may enlarge to enormous proportions and may present a nest of greatly dilated, closely packed, interwoven veins which compress the underlying cord at times almost to a point of flatness. On occasion, the varicosities may extend into the cord itself, and the nutrition of this structure becomes so impaired that areas of softening result. Hadlich [9] reported such a condition in a dwarf who died after cesarean section. At autopsy, an angioma of the pia was found in the lumbar region, in which the vessels invaded the cord and caused it to be markedly distorted. In Berenbruch's [10] case, postmortem examination revealed a cavernous plexus of veins running outside the spinal canal and connecting with a similar intraspinal mass through the intervertebral foramina.

Spontaneous rupture is prone to occur and is cited as the most common cause of sudden paraplegia. The rupture may be free into the spinal canal, as occurred in Harman and Balk's [11] case, which at autopsy

8. Raymond and Cestan: Rev. neurol. **12**:457, 1904.

9. Hadlich: Ein Fall von Tumor cavernosus des Rückenmarks mit besonderer Berücksichtigung der neueren Theorien über die Genese des Cavernoms, Virchows Arch. f. path. Anat. u. Physiol. **172**:429, 1903.

10. Berenbruch, K.: Ein Fall von multiplen Angiolipomen kombiniert mit einem Angiom der Rückenmarkes, Inaug. Diss., Tübingen, 1890, p. 24.

11. Harman and Balk: A Case of Angioma of the Spinal Cord, Brit. M. J. **2**:1707, 1900.

showed a large blood clot in the canal overlying the lumbar cord; or it may be intramedullary, as in the case of Lorenz,[12] in which postmortem examination revealed an angioma the size of a cherry at the seventh cervical segment with hemorrhage into the subarachnoid space and into the substance of the cord as well. Death resulted three days after the time of rupture. It is not unlikely that similar hemorrhages have occurred in other cases and have given rise to the so-called "attacks" of paralysis which are so frequently observed and in which the paralysis clears up to a certain extent as the clot absorbs. Naturally, recovery would be less likely to occur if the hemorrhage had invaded the substance of the cord than if it were free in the canal. Thrombosis is perhaps more common than spontaneous hemorrhage. It has been reported in a number of instances and may affect the vessels within or without the cord proper. In the first instance, it would seem that irrecoverable softening would occur, and even if the thrombus lies outside the cord its effect would probably affect this structure in a similar way. In one of our cases a large thrombus occupied the middle of the hemangioma, and Sargent reports a similar observation. Not only may the pial veins and those of the cord be involved but also others as well. Raymond and Cestan report that in their case the vessels penetrating the cord were tortuous, varicose and aneurysmal, and that some were as large as a quill. In certain instances the blood supply to the surrounding vertebrae may be greatly increased, and the veins of the adjacent soft tissues dilated almost to angiomatous proportions. Perman[13] reports a case of angiomatous changes in the body of a vertebra. Again the epidural veins may be the seat of angiomatous change, while those of the cord proper are not affected. This was found in Hille's[14] case and in one of my own. Jumentié and Valensi[15] report a similar condition in a man of 40 who had had a flaccid paraplegia for three years. When the canal was entered, a mass of turgid tortuous veins protruded extradurally. They were described as resembling a varicocele. It was estimated that this venous plexus extended from the sixth cervical to the seventh dorsal segments of the cord, and it was described as surrounding the roots of the posterior ganglia in this region. Elsberg[16] likewise removed a vascular extradural angioma situated at the fifth

12. Lorenz: Cavernoses Angiom des Rückenmarks, Tötliche Blutung, Inaug. Diss., Jena, 1901, p. 37.

13. Perman, Einar: On Haemangiomata of the Spinal Column, Acta. Chir. Scandinav. **61**:91 (Oct. 19) 1926.

14. Hille, Karl: Hemangiom des Wirbelkanals, München. med. Wchnschr. **71**:1241, 1924.

15. Jumentié and Valensi: Dilatations variqueuses des veines spinales postérieures, Rev. neurol. **19**:81, 1911.

16. Elsberg, C. A.: Tumors of the Spinal Cord, New York, Paul B. Hoeber, Inc., 1925, p. 203-205.

and sixth dorsal segments. It was thought to be a tumor of the cord until microscopic examination revealed its true nature. This is the only instance of successful removal of an angioma that I have found; it was different in nature from the more diffuse lesions under consideration here.

At various operations, the hemangioma has been exposed, and in the majority of instances it was not interfered with. Indeed, it is improbable that such a mass of serpentine vessels could be removed because of technical difficulties, and furthermore it would be unwise because of degenerative changes that would occur within the spinal cord. Frazier succeeded in ligating the largest vessel in his case, but in doing so it was torn, and troublesome hemorrhage ensued. He states that "intervention was limited to ligation of only one vessel, and even this caused temporary impairment of function." Krause [17] ligated a number of vessels in his case and after death, which occurred three months later, only remnants of tissue from the spinal cord were found at the site of operation. From this he points out the danger of tying off vessels. Sargent ligated four vessels in one of his cases but noted "there had evidently been some thrombosis in the mass of veins" before and stated that the bladder and legs were made worse for six weeks following operation when the patient began to improve and finally got back to the condition that existed before operation. In the majority of cases the dura was left open, allowing the mass of vessels to protrude outward and relieving pressure on the cord. It is doubtful if any actual benefit follows such a procedure, although Sargent reports temporary improvement in two cases, and in his third states the "symptoms continued to vary from time to time and on the whole it is doubtful whether much benefit had resulted from the operation." Of six patients operated on, Elsberg reported improvement in four, considerable improvement in one and complete recovery in one after three months. This was the case in which the angioma resembled a tumor of the spinal cord, was extradural in location and was completely removed. Of three patients operated on, Adson reported only one as "improved but not at work"; one was unimproved, and one died.

One symptom is sometimes encountered in physical examination which will give a clue to the diagnosis. In Cobb's case, heretofore reviewed, and in one of my own, it has been present. A nevus or multiple nevi of the skin may occur in the dermatome supplied by the cord segment from which the pial hemangioma arises. This nevus is usually situated on the back, and when present is deeply pigmented—almost black, in fact. It was noticed in Cobb's case, and the presumptive diag-

17. Krause, Fedor: Surgery of the Brain and Spinal Cord, Amer. Trans. New York, 1912, vol. 3, p. 1129.

nosis of "a congenital lesion pressing upon the cord, either a dermoid or angioma" was suggested by Cushing [18] before operation. In a former communication, he had described similar pathologic changes in which facial nevi were found to be associated with vascular tumors of the cerebral meninges. The case reported here is the second in which a nevus of the skin of the back was noted. As I had had the privilege of studying Cobb's case twelve years before, it suggested to me the possibility of a hemangioma of the cord. At least it would seem, when a patient presents signs and symptoms of compression of the spinal cord at a given level and when a nevus of the skin is found in the neighborhood of that level, that hemangioma of the cord may be suspected.

Studies of the spinal fluid have been carried out in several cases. Blahd's [19] case—a typical low dorsal hemangioma—showed clear spinal fluid, under moderate pressure. It contained eleven cells per cubic millimeter, gave a positive Nonne test, a colloidal gold curve of 1342000-000 and a negative Wassermann reaction. One of Sargent's cases revealed a yellow cerebrospinal fluid and no block to Queckenstedt's test. A similar condition was present in my second case. Free blood in the spinal fluid has not been reported at lumbar puncture. It would seem that there might be considerable risk entailed in carrying out spinal punctures in these cases. If the spinal needle should happen to pierce a large vein, serious, perhaps fatal, hemorrhage might occur. Again the lessening of pressure below a hemangioma might be enough to cause rupture of delicate vessels and to result in hemorrhage. In my first case it was thought that rupture of the extradural venous plexus occurred when the dura was opened, and that the escaping fluid allowed this membrane to drop away from the inner wall of the spinal canal.

REPORT OF CASES

Case 1.—*Gradually increasing motor and sensory symptoms for a period of nine months pointing to a lesion at the third sacral segment of the cord. Large extradural plexus of varicose veins found at operation. Improvement.*

History.—A. D., a nurse, aged 28, who was referred by Dr. Gustav F. Boehme on Oct. 30, 1923, had noticed pain in the back of the right thigh in February, 1923. She fell on the floor on two occasions about this time, but did not think it had anything to do with the pain. In May, 1923, the pain became worse. She noticed a stinging sensation in the entire leg, especially while swimming. These sensations would last five minutes, and the leg would draw up when she tried to walk. In June, 1923, the pain became more severe with a knotting sensation in the right thigh and calf, which became so extreme that she could walk only a short distance. She also had severe pain in the lower back, and obtained relief only by lying on the back on the bed and letting the

18. Cushing, Harvey: Cases of Spontaneous Intracranial Hemorrhage Associated with Trigeminal Nevi, J. A. M. A. **47**:178 (July 21) 1906.

19. Blahd, M. E.: Hemangioma of the Spinal Cord, J. A. M. A. **80**:1452, (May 19) 1923.

feet rest on the floor. During the summer she was better, but in the fall the pain became severe in the back and the right thigh. She could not stand up straight, but drew over to the right side. The pain was worse in the morning and after lying down. In October, 1925, while combing her hair, she sneezed and the pain became excruciating in the back and left thigh. She had to lie down; the left leg pained her all day and that night she first noticed numbness in the right thigh. The legs became weaker, and she could not dance or stand on the toes. Once when lying down, she was afraid that she would become paralyzed if she stayed there so she got up again. She said she was "not going to get paralyzed lying down." She was beginning to have trouble in urinating and soon developed complete retention. She also had great trouble with bowel movements; whenever she strained at stool it caused terrific pain in the back, rectum and both thighs. The numbness of the right buttock increased, and numbness of the left buttock set in.

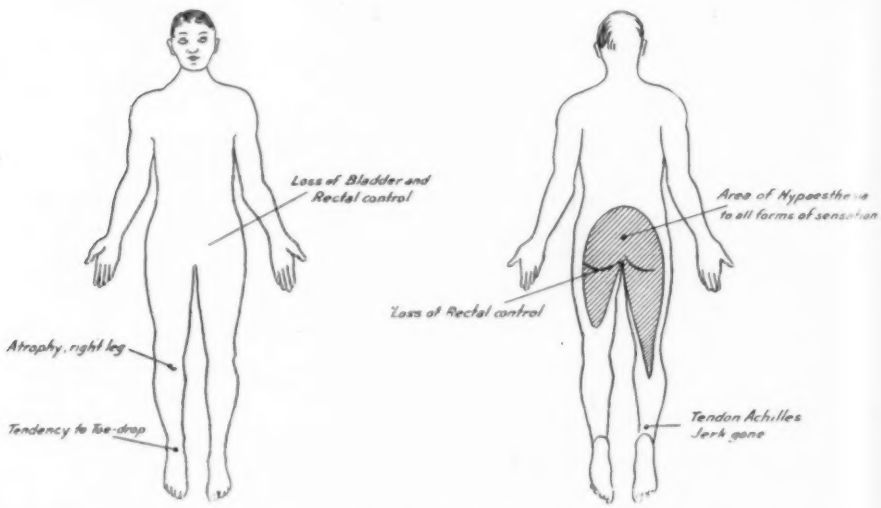

Fig. 1 (case 1).—Area of hypesthesia of the saddle or riding breeches type.

Examination of the Spinal Cord.—Motor: There was no definite paralysis of any muscle group, although both legs were weak; the right ankle was much weaker than the left. There was moderate atrophy of the right calf, with a tendency to toe-drop on the right.

Sensory: There was a definite area of hypesthesia of the saddle or riding breeches type for all forms of sensation. On the right, it extended down to the middle of the calf and on the left to the middle of the thigh (fig. 1). There was a feeling as of cold water running down the right thigh. Muscle, joint and vibratory senses were intact on both sides.

Reflexes: Abdominal and epigastric reflexes were present and equal on the two sides. Biceps, triceps, radial and finger reflexes were active and equal. The knee reflexes were active and about equal. The right achilles reflex was absent, the left present. No Babinski, Oppenheim or Gordon signs, and no ankle clonus were obtained on either side.

Sphincters: The feces were incontinent, and there was complete urinary retention.

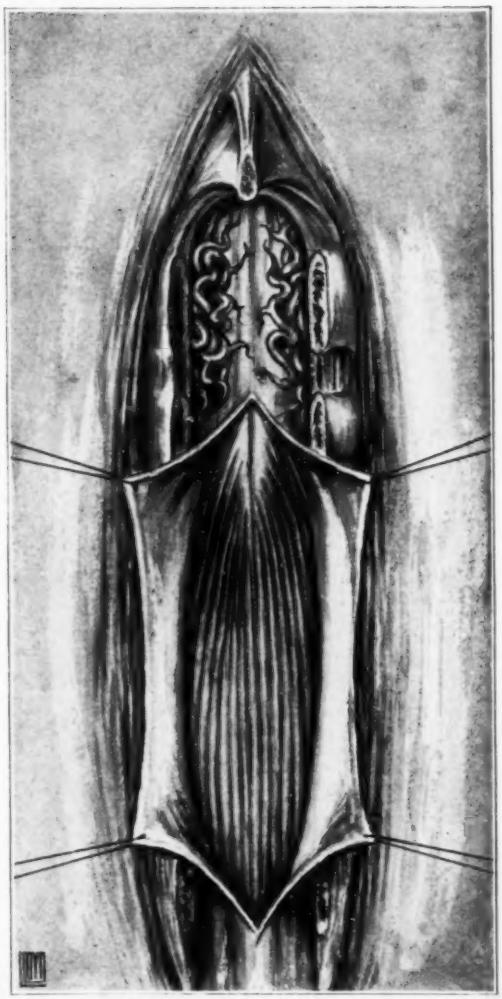

Fig. 2 (case 1).—Extradural plexus of greatly enlarged veins at the level of the cauda equina. Opening of the dura with consequent relief of pressure in the spinal canal is thought to have caused rupture of these veins. Improvement occurred.

Vasomotor: No vasomotor disturbance was present.

Roentgen-Ray: Slight scoliosis was present at the second and third lumbar vertebrae.

Impression.—It was thought that a lesion of the third, fourth and fifth sacral segments was present—most likely a tumor of the cord.

Operation.—Laminectomy was performed Nov. 8, 1923, on the twelfth dorsal to the second lumbar vetebra. The epidural fat was normal. The dura was slightly bluish and under considerably increased tension. The dura was opened by pin prick, and clear spinal fluid spurted several inches. The opening was then slightly enlarged with a knife protected by a grooved director. A sudden gush of clear fluid was followed immediately by a large venous hemorrhage. This was beyond control for several minutes but was finally checked by large cotton packs, which were carefully removed and replaced by muscle grafts. On looking over the field, a large varix of veins was seen lying extradurally on the sides of the cord and extending anteriorly. It was thought that the sudden removal of pressure by the escape of spinal fluid caused these veins to rupture and accounted for the hemorrhage (fig. 2). A few days later the wound was reopened and the dura was opened more widely, exposing the tip of the cord, conus and part of the cauda equina. No tumor was found, but the large plexus of veins noted before was again seen and let alone. Operative recovery was satisfactory.

Postoperative Course.—Dec. 27, 1923: The saddle area persisted to the back of the knee on the right and to the gluteal fold on the left. Motor function was good, but the patient could not stand on tip-toes or on either foot alone. The right achilles jerk was still absent. The patient could now control the bladder and rectum; there was returning sensation in the genitalia, urethra and rectum. There was now no pain in the back, but some pain in the right leg, not so severe as before, was present.

May 16, 1924: The anesthetic area was the same as six months before, except that the area for hot and cold was smaller than that for pain. Practically all motor function had returned. The right ankle would become weak after the patient walked for about a mile; there was no limp. Both achilles reflexes were now present, but the right was weaker than the left. The sphincters were now under control. The patient could stand tip-toe on either foot alone.

December, 1925: The saddle anesthesia alone persisted.

December, 1926: The saddle anesthesia still persisted. The patient was working as a nurse.

Case 2.—Slowly progressive weakness of the left leg for two and a half years followed by sudden paraplegia. Lesion at second lumbar segment. Large hemangioma of cord found at operation. No subsequent improvement.

History.—J. L., a violinist, aged 30, referred by Dr. S. D. Ingham on Jan. 6, 1926, two and a half years previously had first noticed pain in the left foot and leg. He used arch supports at first. Later, he noticed weakness in the left leg and during 1924 developed a drop-foot on this side. During 1925, the pain in the left leg and foot increased. The limb was getting weaker, and atrophy of the calf became noticeable. On Dec. 22, 1925, he played a game of baseball, acting as catcher. In trying to reach for a swift ball he fell, sitting down forcibly on the ground. He finished catching the next two or three innings and that evening attended a dance. The next day he felt tired, and his legs were weak and painful. When he awoke on the morning of Dec. 24, 1925, he found that he was unable to

get out of bed. Apparently he had became completely paraplegic during the night. He had complete retention of urine, which changed to incontinence in the next day or two. Control of the bowels was lost. A lumbar puncture revealed xanthochromic fluid which coagulated *en masse*, giving a typical Froin reaction. The fluid contained 7 cells per cubic millimeter, an increased amount of globulin and gave a negative Wassermann reaction; from 20 to 30 cc. of fluid was removed and air was injected, but no definite level of the column was seen either in the lumbar region or in the sacral canal.

Examination of the Spinal Cord.—Motor: There was practically complete flaccid paralysis of both lower extremities. The man could barely move the toes of the right foot at times.

Sensory: There was complete loss of all forms of sensation corresponding with the second, third and fourth lumbar and all sacral dermatomes (fig. 3). Testicular sense was low. The patient did not appreciate the passage of urine or feces.

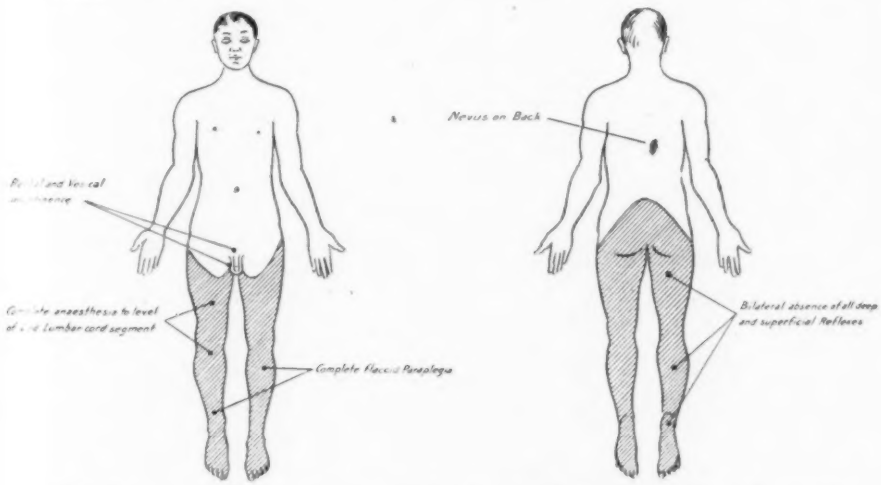

Fig. 3 (case 2).—Area of hypesthesia, showing loss of all forms of sensation corresponding with the second, third and fourth lumbar and all sacral dermatomes.

Reflexes: The abdominal and epigastric reflexes were present and equal. The knee and achilles reflexes were not obtained on either side. There was no plantar response of any sort and no ankle clonus on either side.

Sphincters: Incontinence of rectal and vesical sphincters was present.

Trophic: There was a large sacral burn from an electric pad. A dark pigmented patch, 5 by 3 cm. in diameter, was noted on the right side of the back at about the level of the angle of the scapula.

Impression.—The condition was thought to be a tumor or cyst of the spinal cord at the level of the second lumbar segment. In view of the nevus of the skin, the possibility of an hemangioma of the cord was considered.

Operation.—Laminectomy was performed, Jan. 9, 1926, from the eleventh dorsal to the second lumbar vertebrae. The epidural fat was normal. The dura was bulging but of normal color; it was opened without injury to the arachnoid, which was found to be bluish and thicker than normal. The arachnoid was

opened with a needle and clear spinal fluid escaped, allowing this membrane to collapse and fall back on a massive nest of veins. The picture was one of a packed mass of large tortuous veins. At the center of the field was one especially large vessel, which was thrombosed and almost as hard as stone. The mass of veins occupied the entire field, and the upper margin of the angioma was not determined. One of the large veins from above was ligated and one from below

Fig. 4. (case 2).—Cord structures entirely obliterated by the mass of veins, showing one large thrombotic vein in the center of the field and the two veins that were tied at operation. No improvement occurred.

was doubly ligated and cut (fig. 4). Attempts to ligate other veins did not seem permissible. At no time were any of the structures of the spinal cord or cauda equina seen, as they lay entirely in front of the varix.

Course.—The condition has remained practically stationary. The bladder has become automatic. There has been a little return in the power of the adductors only. One course of deep radiation, given shortly after the operation, produced no results.

CONCLUSIONS

1. Twenty-one cases of hemangioma of the spinal cord have been reviewed. Most of them are venous in origin, although one or two cases that were arterial in origin have been encountered. Spontaneous hemorrhages or thrombosis are likely to occur, with sudden increase in the symptoms.

2. The presence of a nevus of the skin in the neighborhood of the dermatomes supplied by the upper segments of the lesion of the cord should give rise to the suspicion of hemangioma of the cord.

3. Surgery offers little if any hope of improvement in these cases.

PERONEAL FORM OF PROGRESSIVE MUSCULAR ATROPHY

A CLINICAL REPORT OF TWO FAMILIES *

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A controversy has arisen recently between Roussy and Levy on the one hand and Symonds and Shaw on the other, over the classification of two groups of cases which these observers have studied. Roussy and Levy [1] reported the occurrence of a disease in seven members of one family, which was characterized by an acquired bilateral pes cavus, together with total abolition of the tendon reflexes and in some of the cases atrophic weakness of the hands. This, they claim, is a clinical picture hitherto undescribed, and should be regarded as distinct on the one hand from Friedreich's disease, and on the other hand from the so-called peroneal type of progressive muscular atrophy originally described by Charcot and Marie and in the same year by Tooth.

Symonds and Shaw,[2] from a comparison of the details recorded in Roussy and Levy's cases with those found in ten members of a similar family which they have studied and a review of the earliest descriptions of the so-called peroneal form of progressive muscular atrophy, believe that the cases under discussion may fairly be considered as a "forme fruste" of that disease. They also record an instance from another family showing a "forme fruste" and the fully developed disease occurring in two sisters.

In Symonds and Shaw's cases, deformity of the feet was present in all; in six of the patients, the knee and ankle reflexes were absent; in two others, the knee reflexes were diminished and the ankle reflexes were absent; while in the remaining two patients, the right knee reflex was diminished, the left was doubtful, and the ankle reflexes were absent. In the upper extremity the triceps reflex was absent in two, diminished in three others, and normally active in the remaining five; the supinator reflex was absent in six, diminished in two, doubtful in one, and the remaining patient showed a diminished response on the left with a doubtful reaction on the right side.

Localized atrophy in the hands was present in four patients; in two it was present in the legs, while in the remaining four there was no local-

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1. Roussy and Levy: Rev. neurol. **42**:427, 1926.
2. Symonds, C. P., and Shaw, M. E.: Brain **49**:387, 1926.

ized atrophy. The electrical reactions were examined in five of the patients; in two of these, the responses were normal; in another, faradism gave a normal reaction and galvanism was not attempted; the remaining two patients showed a diminished response to galvanism, one in the left peronei and the other in the peronei and anterior tibial muscles.

Our paper is based on the study of two families and shows examples of the "forme fruste" as well as fully developed types of the disease. We, too, have reviewed the literature of the earliest as well as the more recent descriptions of the peroneal form of progressive muscular atrophy; we are inclined to agree with Symonds and Shaw that their cases reported by them, and those of Roussy and Levy, as well as our own, belong to the group of familial heredodegenerative disease originally described by Charcot and Marie, and Tooth.

In our first family, the disease was known to be present in four generations, and members of three of these were available for exami-

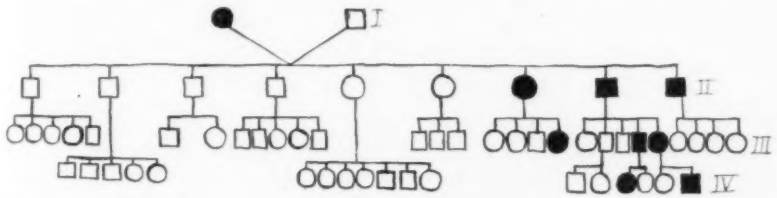

Fig. 1.—Heredity chart 1. Family tree in the first group. The black circles indicate the males affected; the black squares, females affected; the white circles, males unaffected, and the white squares, females unaffected.

nation; in the second family, three generations were known to have had the disease and two generations were examined. In both families, many of the relatives were still in Europe and were therefore unavailable for study. Both families were aware, however, that the condition was hereditary and were able to give reliable data as to the number of instances occurring in the various members of the families.

GROUP 1. THREE CASES SHOWING THE DISEASE IN THE MOTHER, DAUGHTER AND GRANDSON

CASE 1.—*History.*—I. B., a Russian housewife, aged 42, first consulted one of us (M. G.) in 1916, complaining of weakness in both legs and difficulty in gait. The family history relative to the present complaint is shown in figure 1. The patient had been married twenty years and had had three children; the first, a boy, aged 19, was apparently free from the disease; the second died three hours after birth; the third was 6 years of age and showed signs of the condition. The patient's past history had no bearing on the present complaint.

Since early childhood, the patient had known that her feet had been deformed. She was able to get about without difficulty until she was 15; after that, she began to have difficulty in gait on account of increasing deformity in both feet.

She also began to suffer from sharp, shooting pains in both arms and legs, and had a numb sensation in the back with numbness and paresthesia in both legs. About two years after this, she noticed that the hands became weak and numb. She had had no other complaints.

Physical Examination.—In 1916, the fundi and cranial nerves were normal. There was no atrophy, ataxia, weakness, or fibrillary tremors in the upper extremities. The deep reflexes in both arms were present and equally active. The abdominal reflexes were likewise present and equally active. The patient walked with a steppage gait and showed bilateral dropfoot. There was well defined atrophy and weakness in the peroneal and tibial muscles on both sides. The knee and ankle reflexes were absent. No pathologic reflexes were elicited, and there was no ataxia or disturbance in any form of sensation. The electrical response in the peroneal and tibial groups of muscles could not be obtained with the strongest faradic or galvanic currents.

In 1918, she again came under observation and showed, in addition to the previous symptoms, definite atrophy in the thenar and hypothenar eminences of both hands. There was some tremor and a slight deformity in both hands, more marked on the right. The deep reflexes in the left arm were absent, and they could barely be elicited in the right arm. The atrophy in both legs had increased.

Fig. 2 (case 1).—Atrophy of small muscles of both hands and the tendency to claw deformity.

Electrical responses in the arms showed sluggish reactions with both galvanic and faradic currents. The polar relations, however, were not disturbed.

Since that time she had been examined at various intervals and showed a steady but slow progression in most of the physical signs. For the past two months, she had again suffered from shooting pains in the arms and legs. The atrophy in both hands was well marked, and there was a definite claw-like deformity (fig. 2). All the deep reflexes were absent in both the upper and the lower extremities. There was marked atrophy of the muscles of both legs (fig. 3), and there was diminution to pin-prick on both feet below the ankles; all other forms of sensation were intact. There was no Babinski sign, ataxia, nystagmus, or deformity of the spine. The blood Wassermann reaction was negative.

Case 2.—*History.*—I. B., aged 6½, was the youngest son of the patient in case 1. The early development was normal. In the past eighteen months, the mother had noticed that the boy showed a tendency to walk on the front of his feet. He had no apparent functional difficulty in station or gait and had always enjoyed good health.

Physical Examination.—The boy was well developed and well nourished. The gait was normal, with the exception of a tendency to walk on the front of the

feet. The cranial nerves were normal. There was no tremor or ataxia in either the upper or the lower extremities. The deep reflexes in both arms as well as the knee and ankle reflexes were absent. No pathologic reflexes could be elicited. There was slight atrophy in the peroneal and tibial groups of muscles on both sides. Dorsiflexion and abduction were somewhat limited in both feet. Strong and painful galvanic and faradic currents failed to elicit any response in the peroneal and tibial muscles on the left side. Sensory examination showed diminution of pin-prick below the ankles on both feet; all other forms of sensation were intact.

CASE 3.—*History.*—S. F., a Russian housewife, aged 61, mother of the patient in case 1, had no complaints but stated that at the age of 5 some deformity had been noticed in her feet, and that this deformity had become progressively worse in the succeeding years. She was able, however, to do housework moderately well. At the age of 50, she noticed that her hands were becoming deformed. About

Fig. 3 (case 1).—Atrophy in both legs, with bilateral dropfoot.

twelve years previously, she had an attack of severe neuralgic pain in the arms and legs, which lasted several months. About four years previously, she had a similar attack, which lasted five months. Since then she had been fairly comfortable.

Physical Examination.—The woman was well nourished. The cranial nerves were intact. No tremor or ataxia in either the upper or the lower extremities was present. The biceps and triceps reflexes were diminished on both sides; the wrist reflexes, as well as the knee and ankle reflexes, were absent. The abdominal reflexes were present, and no pathologic reflexes could be elicited. Both hands showed well defined atrophy of the thenar and the hypothenar eminences. There was some atrophy in the calves of both legs, and the feet showed a pes equinovalgus deformity. Loss of power in the peroneal and tibial muscles was marked, and powerful faradic and galvanic currents gave only slight and sluggish responses in these muscles. Sensory examination showed diminution of pin-prick sensibility on both feet below the ankles; all other forms of sensation were undisturbed.

GROUP 2. FIVE CASES IN TWO GENERATIONS, A FATHER AND FOUR CHILDREN BEING AFFECTED

CASE 4.—*History.*—I. G., a Russian Jewish tailor, aged 46, whose family history relative to the present condition is shown in figure 4, chart 2, said that his father died of tuberculosis at the age of 52, and that his (the father's) feet were deformed for many years before his death. There was nothing in the past history of the patient relevant to the present complaint. He had seven living children, and there was no history of consanguinity. The present illness dated back to the age of 7, when it was noticed that his feet showed a slight deformity; this deformity became progressively more marked up to the age of 10, and since then had remained stationary. He had never suffered from pains and, except for some fatigue from excessive walking, there had not been any functional incapacity in the legs. About five years previously, he began to have severe pains about the right shoulder and soon thereafter noticed that both arms were becoming weak. The weakness was more marked in the right arm, but it was not severe enough to interfere with his work.

Physical Examination.—The medical examination gave negative results. The gait was a little awkward. The left pupil was slightly larger than the right; both were irregular and reacted more promptly in convergence than they did to light. The fundi showed some pallor in the temporal halves of both disks. The

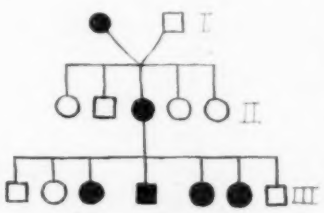

Fig. 4.—Heredity chart 2. Family tree in the second group. The black circles show the males affected; the black squares, the females affected; the white circles, the males unaffected, and the white squares, the females unaffected.

other cranial nerves were intact. There was a fine tremor of both outstretched hands, but no ataxia or other evidence of disturbance in the cerebellar functions. Some atrophy was present in the thenar and hypothenar eminences of both hands. The deep reflexes in both arms were absent. The abdominal reflexes were diminished on the left and absent on the right. There was moderate atrophy in the muscles of the legs and more pronounced atrophy in the small muscles of both feet; the feet showed a pes-equinus deformity (fig. 5*A*). The knee and ankle reflexes were absent, and no pathologic reflexes were present. No nerve tenderness could be elicited, and all forms of sensation were intact. Electrical examination of the muscles of the shoulder girdles, arms, forearms, abdomen and thighs showed the response to the faradic current in these muscles to be much reduced in addition to being slow and vermicular in character; with powerful galvanic stimulation, the response was more prompt. There was no disturbance in the polar relations. In the muscles of the legs and feet, no response was obtained with the most powerful galvanic or faradic currents.

CASE 5.—*History.*—L. G., a bookkeeper, aged 17, complained of increasing deformity of both feet for four years, and pain with twitching in the muscles of both feet for three years. She was the fourth of seven children. One paternal cousin was subject to seizures of grand mal. The patient had suffered from

repeated attacks of tonsillitis. Between the ages of 10 and 14, she suffered from seizures in which she would fall to the ground and lose consciousness for a few minutes; convulsive movements were never noted during these attacks, nor was there a history of tongue biting or loss of control of the bladder or rectal sphincters. Preceding each spell, she suffered from severe headache, which acted as an aura. The menstrual history was normal.

About four years previously, the patient noticed that the arches of the feet were becoming unusually high, especially on the right side. This deformity had become progressively worse since. About one year later, she began to suffer from severe cramplike pains in the legs, which became aggravated when she walked

Fig. 5.—*A*, deformity of feet in case 4; *B*, deformity of feet and atrophy of legs in case 6.

for any great distance. She tired easily. At about the same time, she noticed that the legs and feet felt numb and cold. There were no other complaints.

Physical Examination.—The gait was somewhat awkward. The right pupil was slightly larger than the left; both were irregular, and reacted less to light than they did in convergence. The fundi and other cranial nerves were normal. The outstretched hands showed a fine tremor and a slight manus cavus deformity (fig. 6). There was no ataxia or other evidence of disturbance in cerebellar function. The deep reflexes in both arms were absent. The abdominal reflexes were absent. The muscles of the calves and the small muscles of both feet showed well defined atrophy, which gave rise to an equinovarus deformity (fig. 7). The knee and ankle reflexes were absent. No pathologic reflexes were

elicited. The nerve trunks were not tender to pressure. Sensory examination showed some diminution to pin-prick and touch on the dorsal and plantar surfaces of both feet; all other forms of sensation were intact. Electrical examination of all muscles of the upper and lower extremities showed responses similar to those noted in case 1, with the exception that responses were obtained in the muscles of the legs and feet. The blood Wassermann reaction was negative.

Examination of the remaining members of the family, with the exception of the oldest daughter, had been made and the following additional data have been obtained: The mother and the second and seventh child were found normal. The remaining three children showed the following abnormalities:

Case 6.—*History.*—T. G., a school boy, aged 10, was the sixth child. The only important fact in his past history was the sudden development of internal strabismus in the left eye at the age of 18 months. The only complaint was that he could not run as well as his companions and that when he ran or walked he became easily fatigued. His mother noticed, at the age of 8, that his feet became deformed, and that this deformity had become progressively more marked since.

Fig. 6 (case 5).—Atrophy of small muscles of both hands, with a tendency to manus cavus deformity on the right side.

Physical Examination.—The boy was well nourished. The medical examination gave negative results. Neurologically, he showed marked palsy of the left external rectus muscle. The pupils and cranial nerves were normal. There was a coarse tremor of both outstretched hands, but no ataxia or other evidence of disturbance in cerebellar function. The deep reflexes in both arms could be elicited with difficulty. The abdominal reflexes were present but were more active on the left. The peronei and the muscles of both feet showed moderate atrophy, giving rise to an equinovarus deformity in both feet (fig. 5 *B*). The knee reflexes were elicited only with reinforcement. No pathologic reflexes were present. All forms of sensation were intact. The electrical responses were similar to those described in case 1.

Case 7.—*History.*—L. G., a school boy, aged 14, was the third child in the family. He did not complain of illness.

Physical Examination.—The left pupil was larger than the right; both were regular and reacted promptly to light and in convergence. The fundi and other cranial nerves were normal. He showed a tremor of the hands with a tendency to a manus cavus deformity. The deep reflexes in both arms were present but were difficult to elicit. There was no ataxia or other evidence

of disturbance of cerebellar function. The abdominal reflexes were active. There was no evident atrophy in the lower extremities, but dorsiflexion of the left foot was much weaker than of the right. The longitudinal arches of both feet were abnormally high. The left knee and ankle reflexes were greatly diminished, while on the right, they could be elicited only with reinforcement. No pathologic reflexes were elicited, and all forms of sensation were intact.

CASE 8.—*History.*—P. G., a law student, aged 20, the fifth child, did not complain.

Physical Examination.—The left pupil was larger than the right; both reacted well to light and in convergence. The fundi and the other cranial nerves were intact. The upper extremities did not show any tremor, ataxia, or other evi-

Fig. 7 (case 5).—Deformity of feet, with atrophy of the legs.

dence pointing to disturbance in cerebellar function. The deep reflexes were difficult to elicit in both arms. The abdominal reflexes were present and equally active. The left knee was diminished, while the right could be elicited only with reinforcement; both ankle reflexes were barely present. There were no pathologic reflexes. All forms of sensation were intact. There was no apparent atrophy in the muscles of the calves.

SUMMARY OF THE CASE REPORTS

Two families are reported, one showing four and the other three generations affected with a disease characterized clinically by: (1) equinovarus deformity of the feet (2) atrophy in the calves and small muscles of the feet; (3) onset of the disease in the second half of the

first decade of life, which progresses slowly for a few years, but produces little functional disability; (4) involvement of the small muscles of the hands at a much later period; (5) marked diminution or loss of the deep reflexes in both the upper and the lower extremities; (6) electrical changes consisting of marked reduction in faradic irritability in the muscles throughout the body, but a more prompt response to galvanic stimulation. In addition to the signs described, one family showed disturbances in the abdominal reflexes, and some members had unequal pupils which reacted less completely to light than they did in convergence.

These cases undoubtedly belong in the group of hereditary familial diseases. They resemble closest the clinical descriptions of the peroneal form of progressive muscular atrophy described by Charcot and Marie and independently by Tooth, in 1886.

The characteristics of the conditions, as originally described by Charcot and Marie,[3] are: (1) slowly progressive muscular atrophy beginning in the feet and legs and not involving the hands and arms until several years later; (2) relative integrity of the axial groups of muscles, or at least much longer preservation of these, than the muscles of the distal parts of the extremity; (3) integrity of the muscles of the face, shoulders and trunk; (4) fibrillary contractions in the muscles undergoing atrophy; (5) vasomotor disturbances in the segments of the limbs which have atrophied; (6) usually intact sensations, though sometimes they may be affected; (7) the absence of pronounced contractures in the tendons; (8) electrical changes in the muscles undergoing atrophy; (9) hereditary and familial tendencies.

Tooth's conclusions,[4] drawn from five cases of his own and the reports of those he was able to collect from the literature, vary only a little from those reached by Charcot and Marie. He believed from his pathologic studies, as well as on clinical grounds, that he was justified in regarding the disease as one affecting chiefly the peripheral nerves. In the thirty-nine cases forming the basis of his thesis, thirty-six patients showed the first signs of the disease before they reached the twentieth year of life. In our own patients, in whom the atrophy was at all pronounced, practically all showed evidence of the disease before the tenth year.

The literature contains papers reporting many instances of the occurrence of typical, atypical and abortive forms of the disease. The heredofamilial character has been emphasized by Herringham, Stiefler and others, and recently by Macklin and Bowman. Herringham[5]

3. Charcot and Marie: Rev. de méd. **6**:97, 1886.
4. Tooth: Cambridge Thesis, 1886.
5. Herringham: Brain **11**:230, 1888.

reported twenty instances in a family containing ninety members that he had been able to trace over five generations. In this family only males were affected, although the disease was transmitted by females. Stiefler [6] reported nineteen instances of the disease in a family of fifty-six members in four generations. Both males and females were affected and transmitted the disease. Macklin and Bowman,[7] in a recent article, reported twenty-one instances of the disease among 101 members of a family in five generations. There were eleven females and ten males. Transmission occurred through both males and females. Fifty per cent of the offspring of affected parents showed the disease. No instance of the condition was noted in any of the children of parents who did not show the disease. In regard to the mechanism of inheritance, Macklin and Bowman concluded that in this family the disease was due to the presence of a unit character, and that it was dominant and not self-linked. In our first family there are fifty-seven members in four generations. Up to the present, eight instances of the disease have occurred among them; four males and four females were affected. Only when the parents showed the disease were the children also affected. In our second group, a complete study of the family could not be carried out on account of lack of interest shown by the patients and their parents. In this family, both males and females show the disease. Transmission has been through the males; the one female who shows the disease is still unmarried. They also show, as Macklin and Bowman's figures indicated, that the disease tends to increase with each succeeding generation.

The disease usually begins in the second half of the first decade of life, and the course and its evolution extend over a considerable period of time. In Macklin and Bowman's cases, the age of onset varied between 12 and 40 years. In one of Stiefler's patients, the onset was said to be at 68 years. In all of the patients in both of our groups, when definite atrophy was evident, the onset was noted to be in the second half of the first decade of life.

The protracted course of the disease has been noted by most observers. Spiller [8] reports a case in which the disease was present for at least forty-five years. In one of Warrington's [9] patients, it was present for forty-nine years; in one of Macklin and Bowman's patients, forty-eight years, and another was still alive forty-four years after the

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6. Stiefler: Ztschr. f. Heilk. **27**:219, 1906.

7. Macklin, M. T., and Bowman, J. T.: Inheritance of Peroneal Atrophy, J. A. M. A. **86**:613 (Feb. 27) 1926.

8. Spiller: J. Nerv. & Ment. Dis. **34**:15, 1907.

9. Warrington: Lancet **2**:1574, 1901.

onset of the illness. In our group, one patient has had the disease for fifty-three years, and another for thirty-nine years. Both of these patients have been able to carry on their work without much interruption, despite the disability. The psychic reaction to the disease in all of our patients has been remarkable. As long as the disability did not interfere with the ordinary occupations, they considered themselves well and looked on the deformity as a natural characteristic of their families.

The atrophy of the muscles usually begins in the peronei, the extensor communis digitorium, or the small muscles of the foot. It is only after an interval of years that the small muscles of the hand become affected. In typical cases, the atrophy is limited to the peripheral portions of the extremity and spares the proximal and the axial groups. Sachs [10] reports two patients with atrophy in the infraspinati muscles. His cases were also unusual in that the knee and ankle reflexes were retained; in one there was, in addition, marked scoliosis. In one half of Stieffler's [6] patients, the atrophy began in the distal portion of the lower extremities, and an interval of about twelve years elapsed before the hands became involved. In one of his patients, a woman aged 68, the atrophy began in the hands and the legs were still unaffected at the age of 76. In one of Hatch's [11] three patients, there was atrophy with fibrillary tremors in the pectoral muscles. This author reports that the spinal fluid in his cases showed an increase in albumin and globulin content and that the colloidal gold reaction produced a curve similar to that obtained in cerebrospinal syphilis, resembling most closely that seen in tabes. The blood and spinal fluid Wassermann reactions were negative. Siemmerling's [12] patient showed diffuse atrophy of all muscles of the trunk and extremities, and in addition presented depression, which he classed as melancholia. Pathologically, this case showed degeneration of the posterior and lateral columns, atrophy of the cells of the anterior horn and of Clark's column, and degeneration of the anterior roots, posterior ganglia, and the peripheral nerves. The lack of an hereditary element in the case, with the extensive involvement in the muscles, casts some doubt as to the precise classification of this case. In one of Hoffman's [13] cases, the tibialis and the muscles of the calves were involved and the peronei were spared; in another one of his patients, a lumbosacral kyphosis developed late in the disease, and the clinical picture was much like that seen in muscular dystrophy.

Objective sensory disturbances may or may not be present. When they are present, the sensory changes are usually found on the peripheral

10. Sachs: Brain **12**:447, 1889.
11. Hatch: Boston M. and S. J. **2**:393, 1915.
12. Siemmerling: Arch. f. Psychiat. **31**:105, 1899.
13. Hoffman: Arch. f. Psychiat. **20**:560, 1889.

parts of the extremities, and usually affect pain, temperature and touch. In one of Hoffman's [13] patients, there was disturbance in thermal sensibility. In our cases, the three patients in the first group and L. G. in the second group showed mild sensory disturbances in both feet. Touch and pin-prick sensations were diminished in the first three, and were more profoundly affected in the fourth patient. All other forms of sensibility were preserved.

Electrical changes in the muscles are reported in most of the patients. The faradic irritability of the muscles is greatly diminished; with the galvanic current, the diminution may be less marked, but a partial and even complete reaction of degeneration has been observed in some cases. The electrical changes are, as a rule, widespread and may sometimes be present in distant muscles even when there is no apparent atrophy.[14] In all of the first group and in three of the second group, when we were given the opportunity to test these reactions, we found this widespread diminution in the response of the muscles to both the galvanic and the faradic currents; in one patient, case 4, we were unable to produce any response in any of the muscles below the knees with the most powerful galvanic or faradic currents.

Other unusual features that were present in our second group of patients are the changes in the abdominal reflexes and the anisocoria, with the sluggish response of the pupils to light in case 4 and in case 5. The only reference to alteration of the abdominal reflexes that we could find was in Stiefler's paper; [6] in one patient, he noted that the abdominal reflexes were exhaustible. In one of our patients, the abdominal reflexes were absent; in two others they were unequal and greatly diminished. We were inclined to explain this disturbance of the abdominal reflexes on the basis of interference with the peripheral reflex arc, as there was no other indication of involvement of the pyramidal system at any other point.

The ocular manifestations in this disease have been studied by Krauss,[15] who comments on the paucity of reports concerning them in the literature. He found pupillary changes mentioned four times; optic atrophy has also been noted in about the same number of cases. The patient he reported showed optic atrophy. He agreed with most authors that the characteristic ocular symptom is atrophy of the optic nerves. Four of our patients showed inequality of the pupils, with a more complete response in convergence than there was to light stimulation in two of them.

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14. Oppenheim: Textbook on Neurology, transl., Bruce, London, T. N. Foulis, 1910, p. 253.

15. Krauss: Ztschr. f. Augenh., 1906.

CONCLUSIONS

1. These cases undoubtedly belong to the group of familial heredodegenerative diseases.

2. The absence of ataxia, nystagmus, disturbance in speech, scoliosis and the benign course of the disease speaks against the probability of the cases being abortive forms of Friedreich's disease.

3. The entire clinical picture fits in best with that described by Charcot, Marie and Tooth as the peroneal form of progressive muscular atrophy.

4. These cases also furnish additional evidence to substantiate the contention of Symonds and Shaw that the cases described by Roussy and Levy belong in the same group.

5. Among our patients, instances of "forme fruste" and fully developed examples of this disease are evident.

BRITISH AMERICAN NEUROLOGIC MEETING

SOME IMPRESSIONS

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ST. LOUIS

On July 25, members of the American Neurological Association met with the section of neurology of the Royal Society of Medicine, at London, under the chairmanship of Sir James Purves-Stewart. A series of papers, discussions and case demonstrations carried the program through three days. Late in the afternoon of the last day, the Hughlings Jackson lecture was given by Charles L. Dana. The meeting ended with a banquet to which the American neurologists came as invited guests of the section of neurology.

As the papers and discussions are to be printed in full in a special number of *Brain*, this report of the meeting will be concerned with a more or less critical summary of what happened, rather than with a detailed account of the papers presented. It is not necessary to comment on the high average of merit of the majority of the papers, nor is it necessary to mention the fine spirit of mutual consideration and the absence of attempts to compare the merits of the contributions of the Americans with those of the English. If ever a meeting was international in spirit, it was this London meeting. While comment and criticism were frequent, they were good-natured, friendly and appreciative. An almost complete absence of the spirit of rivalry was manifest, as though the problems of neurology were in themselves too important and impressive to admit of anything suggesting a neurologic contest. The meeting, however, gave one an opportunity to observe the English neurologist and to compare him with his American brother as to methods, interests, tendencies and manner of looking at things. That there are interesting differences and that they are, so to speak, national characteristics belonging to the two schools of neurology are common knowledge. It is with comments on these differences and such likenesses, together with the strength and weakness of both neurologic groups, that this paper will mainly deal. The program submitted by the English and American groups can be taken as a kind of estimate of tendencies and tastes, showing, perhaps, lines of future development and growth. I, at least, experienced a growing sense of pleasure when this London conference appeared to show that a distinctive American neurology was finally making its appearance—an outgrowth of the English school in many ways and in many of its characteristics, yet showing certain native points of departure and certain highly interesting individual developments. It

is well if this is true, for neurology, even if in its essence it is without specific national affiliations, must meet national demands and must see its own particular task close up.

The data for such a comparison and the observations on which these comments are based are drawn not only from the papers presented but also from the many bits of conversation and the many frank discussions. Many of these occurred apart from the meetings, often most enjoyably at the many dinners and luncheons arranged through the hospitality of the London neurologists. That part of the London experience is pleasantly fixed in the minds of all who shared it.

The first day's program consisted of short papers contributed by each group. As a rule, the reading of the papers took about twenty minutes, and they were concerned with such subjects as might be regarded as of neurologic interest and importance. In some instances the special points were illustrated by case reports, and occasionally actual experimental evidence was offered. This first day's program was of a kind that might, with few exceptions, have been presented in any neurologic section of a general society. The predilection of the English neurologist for detailed case reports, well arranged and commendably written, but often rather diffuse in relation to the special point to be emphasized, was evident in several papers. This refers particularly to the papers of the first morning. In listening to the first day's program, one felt that it had been arranged as a sort of feeler, a kind of preparatory gesture to the more important part which was planned for the second day. It is somewhat difficult to single out from the many papers the few that stand out in my memory and those that warrant some special comment. The several that are here mentioned are selected not because of their intrinsic importance, but because they seem to show particular features suggesting national differences and national characteristics.

In this way, Prof. Edward Sharpey Shafer's contribution, "Some Effects of Severance of Cutaneous Nerves," stands out. The experiments on which the conclusions of the paper were based were performed on Professor Shafer's arm; the sensory phenomena of nerve degeneration were studied by him with the most meticulous accuracy, and the resulting responses were set down with the discriminating acumen characteristic of his work. The presentation itself was most interesting, and the deliberate method in both the approach to the investigation and the conclusions finally reached were reminiscent of another English neurologist, Sir Henry Head, who, unfortunately, was prevented by illness from taking part in the meeting. This paper, which will appear in *Brain*, is earnestly recommended to all American neurologists, as is the discussion of it by Wilfred Trotter. Trotter's incisiveness in stating a point of view, as well as the rigorous accuracy of his premises and large

imaginative grasp of the subject, was an example of the best English method in discussion, and stands out as a more than ordinary critical comment. He not only amplified the points brought out in Professor Shafer's paper, but added facts and theories of his own which developed the subject much further than the paper itself did. It seemed to many of us who listened in admiration to Trotter's comments that his discussion was totally unprepared; and the surprise element, so often lacking in carefully prepared discussions, was thus present and added the touch of spontaneity which is always desirable.

The clinical report of Norman Dott, a young Scottish neurologic surgeon, stands out vividly in my memory. It dealt with a patient with hydrocephalus who was cured after surgical treatment by a method which has been described in the literature. The merit and interest in the paper were in the certainty and the careful description of the anatomic problem involved rather than in the originality of the procedure. I was pleased to note the use of the word "cure" in the title, and the energy with which this result was emphasized. I mention Dott also, because he seems to be one of the two or three English or Scottish neurologic surgeons who are pursuing the specialty of neurologic surgery along the same lines as their American surgical colleagues, and who are thus bringing to the attention of English neurologists the necessity for the neurologic surgeon to be a well trained neurologist who is willing to divorce himself from the activities and burdens of general surgery.

An amusing but interesting diversion was furnished by Cornwall's moving pictures of a kitten, demonstrating the loss of many reflex movements as a result of cerebral and cerebellar agenesia. This was a contribution to the subject of animal behavior in relation to environmental demands, the animal studied being congenitally handicapped. The various maneuvers of the animal in attempting to overcome, for the most part unsuccessfully, the various types of stimuli to which it was exposed added a touch of experimental reality to the morning's program. The carefully worked out family history of this animal seemed to amuse and interest the English members of the audience, and comments were made on the ingenuity of the American in furnishing this social service history as a part of the demonstration.

In the afternoon there were a number of other papers of a somewhat technical nature, which suggested that even in so general a program as this, there was room for a more intensive type of contribution. Osnato and Killian's study on the chemical composition of the blood and spinal fluid in epilepsy was typical of the painstaking and thorough effort which is so common in organized laboratory work in America. This work was most meticulous, many chemical procedures being employed. Although the final conclusions were somewhat disappointing, considering the vast amount of work done, the experiments served to show a method

of approach which may yet become useful. The technical application of the chemical procedures was beyond the comprehension of most of the audience, yet there was much interest in awaiting the final conclusions. This paper was succeeded by another technical paper on chemical studies of the formation of the spinal fluid. Evidently, the only feasible method for a solution of this intricate problem is through physiochemic procedures, that is, the study of a closed system of fluid, approached as a problem in physics and chemistry. This paper was discussed by Fremont-Smith, whose intelligent grasp of the problem made it much plainer to the neurologic listener. These two papers seemed to show that for the solution of many of the problems of neurologic mechanics, the same methods must be used that have been so successful in the great research laboratories of physical chemistry. It was also proved that a type of neurologist so trained is essential to progress in problems of this sort, of which there are many. Favill's ingenious diagrams and explanations of the semicircular canals and the muscles of the eye in relation to nystagmus were contributions of real significance. They showed ingenuity and imagination which in many ways, perhaps, are more characteristic of the American than of the English neurologist.

All the papers of the first day were well worth listening to. They were presented clearly and in most instances with commendable brevity. The discussions, for the most part, were disappointing. In America, discussions are almost always negligible. The American Neurological Society seems to have lost the art of discussion, but most of us, through previous experience, had been led to think otherwise of an English meeting. There does not seem to be any explanation for the lack of discussion at this meeting. What happened the first morning became the habit in all the other meetings. The audience appeared to have little to add, and one paper followed the other without important comment, criticism or articulate notice. This was not the case, however, in one of the afternoon meetings at which a number of prepared discussions were given.

The significant contributions of the American neurologists took place on the second day when the cerebellum was the subject for consideration. Some of the material had been presented at the last meeting of the Research Society in New York, but was given in London more completely and compactly. The whole contribution had a certain continuity which it lacked there. The introductory paper on the cerebellum was given by Riley on the comparative morphology of the cerebellum. This was presented by a carefully worked out group of photographs and diagrams, together with those of the animals from which they had been studied. They were an impressive series, and the growth of the cerebellum anatomically and its increasing physiologic importance were clearly demonstrated. Nothing more suggestive of the rôle of the cerebellum in the scheme of things could have been thought of. The

importance of this paper was clearly evident not only because it introduced the subject of the symposium so well, but also because it served to show one phase of the activity of American neurology. It also called to the attention of the English group a type of scientific activity associated with what may well be called the Tilney school, which is, in essence, the emphasis on comparative anatomy as a means of understanding the functions and forms of the nervous system of man. This series of papers was a good illustration of the American method in a massed attack on an intricate and difficult problem. A good deal of experimental material was shown; moving pictures were displayed and photographic slides were abundantly used. There was no summing up and no attempt, apparently, to correlate the results of the intensive effort which this work as a whole demonstrated. It did, however, show that an increasing interest has been awakened toward a physiologic and experimental attitude in neurologic problems, and illustrated, in a rather marked fashion, a departure from the anatomic section-cutting type of research which has so long dominated neurologic programs. The latter form of research has served its purpose. Another kind of inquiry was needed, and this part of the program suggested that the new field has been enthusiastically entered. The vast amount of experimental effort displayed and the variety of results that were garnered made one wonder just how much clarification will result from experimentation on animals, most of which could not be said to present the essential factors in cerebellar function found in man or in animals forced to assume the upright position. Perhaps it is just this point that prevents any general or fundamental conclusions; indeed, this may be one of the strong points in this type of research. It means the gathering of single sets of observations which may finally allow deductions of simple truths. The application of such hard won facts to man, who is subject to defects in his nervous structure, those of the cerebellum being of particular interest in the present discussion, must wait for a broader experimental field and a more inclusive type of investigation. These papers are to be considered as a contribution to the knowledge concerning the mechanism of the reflexes. Those characteristic of the cerebellum and midbrain can be identified and studied in the experimental animal with the cortical control removed by the ingenious methods of Pollock and Davis. Isolated territories of the central nervous system can, in this way, be observed as they react to definite and primitive stimuli. The contributions of Pollock, Davis and Mussen brought out these facts with a surprising degree of clearness, perhaps overaccentuating the simplicity of the problem by the very clarity of the demonstrations. There was no one in the large audience that morning who did not feel, however, the presence of an English physiologist in the background of most of these experiments. His work

had stimulated many of these researches, and his original conceptions of the importance of primitive reflexes formed the foundation on which most of the experiments were based. Sir Charles Sherrington was present at one of the afternoon meetings, but his scientific influence was felt at all of them.

The concluding paper of the second morning's program was Dr. Weisenburg's clinical summary of the whole subject. This made the previous papers more realistic, owing to the possibility, however remote, that out of such work facts may eventually come that will throw light on the diagnosis and handling of cases in which cerebellar function is affected.

On the afternoon of the second day, a series of clinical demonstrations was given by the English neurologists. According to the usual English methods, the patients were assembled in an anteroom together with their case histories and other important data, both clinical and laboratory. There they could be examined at leisure. Those who were presenting the cases were at hand in each instance, and they demonstrated such signs and symptoms as were of importance. In this fashion, the audience of the afternoon meeting was acquainted with the cases to be demonstrated, as the interesting points in any case had been studied and the data personally obtained. When the patients were shown later in a more formal fashion, considerable familiarity with the cases was evident. Many of the cases were intensely interesting, bringing up problems of diagnosis and the interpretation of data obtained by examination, as well as the consideration of various laboratory results. Little discussion followed probably either because there was too little time or because most of us were surfeited with neurologic information, the morning program almost having quelled the controversial spirit.

The third day's program consisted chiefly of the presentation by the English neurologists of a symposium on sensory disorders in organic disease of the nervous system. In the afternoon a group of unrelated clinical or pathologic reports was presented. The scientific program then finished was followed at 5 o'clock by the Hughlings Jackson lecture.

The American neurologic group looked forward to the third morning's session with a deal of anticipation, as both Gordon Holmes and Kinnier Wilson were on the program. Certain of the Americans were asked to discuss more or less formally the presentations of the Englishmen. As I think back on this part of the program, the impression it created on me at the time is strengthened; i. e., that given a specific subject, the English neurologist will, in a general way, present it with the idea of developing, as far as possible in the time allowed, a complete summary embracing aspects that ordinarily escape attention in the American type of presentation. In this sense, both Wilson's and Holmes' reports almost completely covered the subjects of their papers.

A noticeably excellent style and a decidedly interesting narrative quality were characteristic. Wilson's paper was particularly important, because he attempted to correlate paresthetic sensations with definite cerebral localization. It should be read in its entirety and studied with the care that it merits. While the sensory symposium was not nearly as dramatic as the papers on the cerebellum presented by the Americans, there was much food for thought and consideration in this series of papers. The discussions were surprisingly good, on the whole, differing markedly from the discussions of the previous papers. This point has been alluded to before; the reason, no doubt, was that some of the discussions were evidently carefully prepared. This was particularly true of the discussion of Walshe, which was a striking example of careful preparation and critical estimation of important points. It is not possible to compare the symposium on the sensory system with the papers on the cerebellum. They dealt with different subjects, and were written from entirely different points of view. One group of papers dealt strictly with experimental work, and the other group concerned clinical work with physiologic interpretations.

On the afternoon of the last day, short papers on various subjects were presented. The number of papers was entirely too great for the time allotted, and many of them deserved more attention and deliberation than was afforded. Particular notice should be given to the presentation by Adolf Meyer of the late Charles B. Dunlap's work on pathologic changes in Huntington's chorea. Those who knew him either personally or through his work were prepared to hear a most conscientious and carefully worked out series of microscopic studies on the pathologic changes in Huntington's chorea. His is probably the most elaborate piece of work ever done on the subject. It will probably be the final treatise on methods of studying pathologic changes according to Dr. Dunlap's system of investigation. In a general way, it dealt with laborious cell counts of serial sections throughout the whole of the central nervous system of patients with chorea, and contained painstaking clinical observations. One wonders whether this paper is not, perhaps, one of the last of that type of research which represents more the older notion that pathology of nervous diseases can be definitely solved by the enumeration and description of histologic changes.

When the afternoon series of papers was concluded at 5 o'clock, the Hughlings Jackson lecture was given by Dr. Charles L. Dana. Those of us who had read previous Jackson lectures were under the impression that the lectureship was given under formal and elaborate surroundings. Apparently this is not the case. Quite casually, as it were, after the meeting had adjourned and a slight recess was taken, Dr. Dana was informally conducted to the chair which he had so ably filled in many of the meetings, and, in a simple and gracious manner, began reading

his paper. The subject, which was a surprise to most of Dr. Dana's American colleagues, was a sort of historical summary of the growth and development of American neurology, as observed personally by Dana. Nothing, apparently, could have interested the English members of the audience more, especially since few of them were acquainted in any detail with the growth of American neurology. Jelliffe's brilliant historical study of the subject was evidently unknown except to a few. The informality and graciousness of Dr. Dana's presentation seemed to surprise every one; all his hearers, whether English or American, were grateful for a glimpse into the development of American neurology interpreted by one who has become almost a traditional and historic figure in building up the specialty in America. A word of gracious appreciation by Professor Sherrington concluded the scientific program of the meeting.

That evening, the members of the American Neurological Association were the guests of the section of neurology at a banquet at the Café Royal. At least 150 attended. This dinner was a fitting conclusion to three days of neurologic fellowship. The atmosphere of the dinner was so cordial and friendly that the bonds of scientific comradeship were strengthened by personal respect and affection. The after dinner speeches reflected this spirit, and the last speech by Dr. Weisenburg expressed, among other things, the hope that a meeting of this sort could be held in America in the near future. The statement was perhaps more enthusiastically applauded than any of the remarks by the other speakers.

In thinking about the meeting, as a whole, and in attempting to formulate some notion of its benefits, both to the Americans and to the English, the following occurred to me: The distinct difference between American and English neurologists was apparent to any one who observed them closely. In the first place, it was rather remarkable that so many of the papers of the American contingent were presented by comparatively young men, in contrast to to the English contributors, most of whom were of more mature age. The younger group in the American Neurological Association was represented on the program to a large degree, and this seemed to suggest that there is no lack of promising work being done by this group. Almost all their papers showed the tendency, which has been emphasized before, of approaching neurologic problems from the experimental and physiologic aspects. In contrast, the papers presented by the English seemed rather to avoid the experimental phase of neurology. One cannot know whether this was a pure coincidence or not, but the contrast was sufficient to cause more or less comment. The undoubted superiority of the English neurologist in presenting a clinical paper or report, in the arrangement of the material, the style in which it is written and the

narrative quality of the presentation, was striking, although in this particular meeting there was less chance for comparison. In spite of the absence of laboratory experiments, the English neurologist frequently showed the effect of his physiologic insight and training, that is, he attempted to correlate his clinical observations and deductions by means of physiologic interpretations, as though he were trying to make up for the lack of purely experimental data by infiltrating his clinical observations with physiologic methods of analysis. This difference is probably explained by the dissimilarity in the organization of the American and the English neurologic departments, and by the fact that few English hospitals in which neurology has become a separate service have laboratory affiliations that compare with those becoming so prevalent in America. In many instances one felt the separation of the experimental laboratories from the neurologic departments not only because of physical distance but also because the neurologic and laboratory departments did not seem in any way closely joined. Essentially, the English neurologist is a clinician in the older sense of the term; he is also a consultant, not in the American conception of the term, but in the definite organized English interpretation. This would suggest a certain aloofness from the actual problems presented by neurologic cases, from the standpoint of their personal management and a personal and active interest in their progress. This is true, not through any fault of the neurologists themselves, but as a part of the development of consultant specialty in this as in other branches of English medicine. Then, too, in many hospitals the neurologist is expected, and in fact eagerly desires, to keep in touch with the practice of internal medicine by actually taking care, in his own wards, of patients whose condition has no neurologic interpretative phase. While this in many ways broadens the neurologic attitude, it causes diversification of effort and rather lessens the intensity of special investigation. Owing to this, the English method has, perhaps, turned out a more precise type of clinical neurologist in organic diseases of the nervous system than has the American method, but, on the other hand, it has perhaps diminished a certain intensification toward individual aspects of the problem which is so common in American neurology.

A strange factor about the program, as viewed in retrospect, was that papers or investigations which departed in any way from what is called organic neurology were almost entirely lacking. The tremendous field of neuropsychiatry apparently did not find a place on the program. It was as deficient in the phase of neuropsychiatry as were the meetings of the American Neurological Association a decade ago. The only psychiatric paper was submitted by an American, and it stood alone among the welter of organic problems presented for discussion. The group of American neuropsychiatrists which is represented to some extent in the American Neurological Association and occasionally suc-

ceed in procuring places on its program was totally absent from this London meeting. A growing feeling exists, however, among the younger English neurologists, in London, at least, that a change is taking place, and it was interesting to find that there is a group of men who manifest a lively interest in psychiatric problems, as well as in the neuroses and a growing concern for psychologic problems of all sorts.

Neurology in general would be greatly benefited if neurologists of different nationalities were successful in developing characteristic trends in their work. If, for example, the American Neurological Association develops into a society in which every phase of a problem affecting the nervous system can be brought to the attention of the total membership, and if the American type of neurologist developed from this stimulus is equally interested in organic neurology, so called, the psychoses and the intermediary field which lies between them, then an American neurology will come into existence. There is, perhaps, no more interesting and fascinating experience than the one furnished by this combined meeting of the English and American neurologic groups. When the papers are finally assembled, they will furnish a splendid summary of the present neurologic status of the two English speaking nations. Out of the meeting came, it would seem, a better understanding and a finer appreciation of the efforts of both English and American neurologists and an admiration for their leaders, from which will develop even a finer spirit of scientific hospitality than existed before this meeting.

Clinical and Occasional Notes

A CASE OF MULTIPLE SCLEROSIS COMPLICATED BY CANCEROUS METASTASES TO THE SPINE*

JAMES H. HUDDLESON, M.D., NEW YORK

REPORT OF CASE

History.—V. B., aged 48, a dressmaker, when first seen in the outpatient department of the Salpêtrière, May 17, 1924, complained of difficulty in walking, shocks in the lower extremities, and numbness and tingling of the right hand. Except that she had been treated for a long time for anemia, and had had two miscarriages, the past history was without importance; there was no history of infectious disease. The family history was unimportant.

The onset was gradual, and occurred at least eight years before examination, with shocks in the legs that varied from time to time. Four years later, the shocks increased and were supplemented by sensations of heaviness with difficulty in walking. After further remissions and relapses, the patient had been unable for a year to go about without assistance; she complained of great rigidity of the legs, especially after resting for some time, and of an unsteady gait. Six months before the first examination, she began to have numbness and tingling of the right hand and arm; they felt hot and cold by turns, and there was a subjective loss of sensation; she became practically unable to sew or write through inability to hold the needle or the pen. Tingling was also noticed in the left hand. Later, there was dulling of vision with headaches.

Examination.—The gait was unsteady and spastic; the knee reflexes were lively, the ankle reflexes diminished; a Babinski sign was present bilaterally. The abdominal reflexes were abolished. The arm reflexes were normal. No objective loss of sensation was demonstrable, except a slight diminution to the tuning fork over the right leg. Moderate lateral nystagmus and slight anisocoria were found. There were no cerebellar signs and no sphincteric disturbances. The blood pressure was systolic 150, diastolic 90. A vaginal discharge was noted, which cleared up under treatment in three weeks.

Course.—About a month after the first examination, the patient began to complain of violent girdle pains in the flanks. Two months later she entered the hospital, and died after six weeks' residence (Sept. 20, 1924). During this time, the pains were controlled only by morphine.

Examinations in the hospital showed: a trace of albumin in the urine; negative blood Wassermann reaction; normal eyegrounds. The spinal fluid was described as follows: clear; pressure, 44 (seated); albumin (Sicard), 0.4 Gm.; Pandy and Weichbrodt tests, positive; lymphocytes, 3; Wassermann reaction, negative; colloidal benzoin test, 012202221000000. Roentgen-ray examinations showed enlargement of the liver and heart, but nothing abnormal in the spine.

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* From the service of Prof. Georges Guillain, laboratory of Dr. Ivan Bertrand, Hospice de la Salpêtrière, Paris.

Necropsy.—The conditions found were: cancer of the second lumbar vertebra with pachymeningitis; metastases from a cancer of the cervix of the uterus, carried to the mesenteric and mediastinal lymph nodes; pericarditis with effusion; terminal cardiac failure.

The gross specimen of the lumbar spine, sawed longitudinally, showed a mottled appearance of the affected bodies, and an irregularly softened consistency to the touch. The vertebral bodies had remained normal in external form and volume, and there was no compression of the cord. Under the microscope, the neoplastic cells had a fibrosarcomatous morphology. A great part of the metastases were necrotic. The bony trabeculae had not been altered, being neither more nor less numerous than normal. An intervertebral disk was found slightly involved; this was unusual, according to Pétouraud's[1] recent survey of spinal cancer.

A lumbar spinal ganglion showed degenerative changes in the cells, not to a marked degree but sufficient to warrant the conclusion that degeneration had exceeded the normal rate and should be ascribed to a pathologic process; the data are not quantitative but need not be disregarded on that account, according to Bertrand.[2] Enlarged ganglion cells with poorly staining protoplasm, including some of the cogwheel type, were particularly noticeable together with some in which the nuclei were eccentric or extruded.

In the lower dorsal and lumbar spinal nerves, some demyelinization was apparent. Certain of the remaining myelin sheaths were knotted or convoluted in places, in the process of undergoing degeneration. Others showed swelling and breaking up of the myelin. At most, the degeneration was scattered and discrete, not massive.

Extradural cancerous infiltration was seen microscopically at the level of the lower dorsal and upper lumbar roots. Nothing neoplastic was found intradurally (in agreement with Coste's[3] statement that secondary neoplasms have not been reported as penetrating the spinal meninges). The metastatic cells in the tissues about the meninges were embryonic in type.

In the spinal cord in this case, one did not find the ascending degeneration in the posterior columns mentioned by Weil and Kraus[4] as a "frequent complication of cancer metastases to the dura and due to posterior root involvement." The comparatively short duration of root symptoms may account for its absence in this case.

Numerous plaques of sclerosis, old and recent, were found throughout the cord and brain stem. Recent plaques in the white matter showed the partial retention of myelin sheaths described by Guillain and Bertrand.[5] One comparatively small, recent plaque, affecting the substance of Rolando in an upper cervical segment, had eradicated the fibers which normally[6] pass radially through it between the primary and secondary networks.

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1. Pétouraud, C.: Le cancer rachidien, Thèse de Lyon, 1926.

2. Bertrand, I.: Les processus de désintégration nerveuse, Paris, Masson et Cie, 1923, p. 179.

3. Coste, F.: Le cancer vertébral, Thèse de Paris, 1925.

4. Weil, A., and Kraus, W. M.: Cancer and the Spinal Cord, Am. J. M. Sc. **171**:825 (June) 1926.

5. Guillain, G., and Bertrand, I.: Contribution à l'étude histopathologique de la sclérose en plaques, Ann. de méd. **15**:476 (June) 1924.

6. Guillain, G., and Bertrand, I.: Anatomie topographique du système nerveux central, Paris, Masson et Cie, 1926, p. 314.

An old plaque, in the posterior columns of one side at the level of the eleventh dorsal segment, showed the more complete demyelinization characteristic of longer evolution. There was also a markedly undulant and vorticose appearance of the myelin sheaths about the plaque, extending almost through the posterior columns of the opposite side. This appearance of irregularly tangential sectioning, in place of the normal transverse-section appearance, may possibly be ascribed to deformation associated with contraction about a plaque of long standing, or possibly to some unexplained process of attempted regeneration.

A slight degeneration along the pyramidal tract, commonly observed in multiple sclerosis of sufficiently long standing, was traced throughout the cord. However, there were so many plaques scattered among all the tracts that it was difficult to determine a clear cut secondary degeneration of any one tract. Demyelinization in Flechsig's and Gower's tracts was seen, at different levels, adjacent to plaques, but did not extend for any distance beyond them and could not be ascribed to a secondary degeneration.

SUMMARY

A case of multiple sclerosis, beginning in a woman of 40 and developing gradually over a period of eight years, was suddenly complicated by upper lumbar root pains, followed by death twelve weeks later. Metastases from a cancerous uterus were found at autopsy to have involved abdominal lymph nodes and lumbar vertebrae.

Cancerous cells of fibrosarcomatous appearance had invaded the lumbar vertebrae, and similar cells of an embryonic type were present in the tissues surrounding the lower dorsal and upper lumbar nerves at their exit from the spine. Degenerative changes (probably due to pressure from metastatic growths) were visible in a lumber spinal ganglion and in lumbar and lower dorsal spinal nerves; no definite ascending degeneration within the cord, ascribable to these extramedullary degenerations, could be traced in the columns of Goll and Burdach.

The characteristic lesions of multiple sclerosis, old and recent plaques, were found throughout the cord and brain stem. Mild associated secondary degeneration of the pyramidal tracts was present.

The sclerotic and neoplastic disease processes are independent so far as ascertainable. Their interest lies in the unusual circumstance of association in the one case.

Obituaries

THOMAS WILLIAM SALMON, M.D.
1876-1927

An unfortunate drowning accident on Aug. 13, 1927, deprived American psychiatry of one of its most outstanding figures, and American public life of a great physician, teacher and earnest citizen, who for years fought the battle for justice for the most neglected citizens—the insane. What his many friends have lost in his passing may be expressed in feelings, not in words.

Thomas William Salmon was born in Lansingburg, N. Y., on Jan. 6, 1876. His father was a physician in general practice in that neighborhood and his early and late training was always in contact with medical work. At the age of 19, he was left alone in the world owing to the deaths in rapid succession of his younger brother, his mother and his father. He inherited little of worldly goods, but an intelligence, widely varied abilities and the capacity for clear thought and determination that in after life were to lead him to the high positions he honored.

Returning from England after the death of his father, he entered the Albany Medical College. His lot was not easy, for much of what he spent he had to earn. His facile talent for line drawing provided one source of income in the making of illustrations for medical publications. After his graduation, in 1899, at the age of 23, he first took up a country practice in Brewster, N. Y. Shortly after his marriage in December, 1899, he was obliged to relinquish his practice for reasons of health, and, on medical advice, moved to the northern part of the state where he accepted a position on the medical staff of Willard (N. Y.) State Hospital for the Insane. Here he found some field for work and interests which were to be his throughout life and to which he was to be such a signal honor. It was his great love of the sea, which ever was a lure for him, amounting almost to a passion, that led him in 1903 to leave the state hospital service and to take a commission as assistant surgeon in the United States Marine Hospital Service (the present Public Health Service). He thoroughly enjoyed his contact with sailors, and one unforgettable cruise to Labrador, as a surgeon on a revenue cutter, remained ever fresh in his memory. In 1908, he was promoted to the rank of passed assistant surgeon in the Public Health Service, and his great abilities were beginning to be recognized outside of the service. His scientific heart still remained faithful to the duty that he saw—the care of the insane—and in 1911,

T. W. SALMON

on leave of absence from the health service, he was appointed chairman of the New York State Board of Alienists. (The present name "Department of Mental Hygiene" reflects well the striking influence wielded by Dr. Salmon on medical and public thought of this country.)

Dr. Salmon's first great opportunity came in the formation of the National Committee for Mental Hygiene. The idea, then struggling feebly but bravely for recognition, was new, and it appealed strongly to his idealism. In it he saw a mechanism for the carrying out of a life's work worthy of the best of practical idealists—the securing of adequate care for the insane and the possible prevention of the economic and personal waste which is the saddest side of mental disease. In 1912, Dr. Salmon became medical director of the National Committee for Mental Hygiene, again on leave from the Public Health Service. His signal ability as an organizer, which later was to bring him such great prominence in medical organization, here found almost adequate material with which to work. The influence of the committee and the ideal for which it stood grew under his guidance. He early realized that the care and treatment of the mentally sick must first be modernized and raised to a level equal to that accorded to other kinds of sickness before the question of mental hygiene—in the sense of prophylaxis—could be thought of. Methodically, he approached this enormous problem with an energy that came from his unbounded enthusiasm. The surveys he made in North Carolina, and later in Texas, should stand forever as milestones in the path of the progress made in the care of the insane in this country. The governor of one state included Dr. Salmon's recommendations in his platform when he campaigned for reelection. Having blazed the trail, he raised funds for further surveys and selected able, earnest workers to make them. His surveys were the models.

Gradually, but more rapidly than most new ideas progress, mental hygiene attained prominence. Its influence began to be felt, at first in nonmedical circles and later among physicians. Dr. Salmon ably and carefully directed its course toward recognition as a public asset. Many were the disappointments and losses of ground gained by great effort. Age-old notions and deepset prejudices are not to be overcome by simple facts. It took the patience, the determination and the sincerity of a Salmon to modernize the medieval attitude of the public and of most of the profession toward mental disease, in the short space of ten years. Dr. Salmon's work for mental hygiene was interrupted by the war in 1917, and in 1919 he returned to take up the work of the committee. From 1915 to 1921, when he resigned from both, he was on the staff of the Rockefeller Foundation while directing the committee.

The World War gave Dr. Salmon the second and his greatest opportunity to show the gifts with which he was so plentifully endowed and to prove his right to the high regard his fellows so generously

accorded to him. As with everything else, he learned the lesson of the war and foresaw the neuropsychiatric needs of the army more rapidly than most men. Although he was never a dreamer, he could see into the future and divine its needs with accuracy from the experience of the past. He had brought with him from youth his belief in himself and in the rightfulness of his ideals, always unselfish, and, as always, he applied himself to bring them to realization with a measure of success that is denied most men who seek to establish something new. By 1915, Dr. Salmon had developed the major part of his plan to organize a neuropsychiatric service for the army in war, which was later to be hailed as one of the real advances in the military as well as in the humane side of the art of warfare that the World War produced. When he first proposed it, it was dismissed with scant encouragement, but discouragement ever acts only as a spur to the enthusiast who believes he is right.

When the United States entered the War in April, 1917, Dr. Salmon applied for a commission in the army, and was awarded the rank of major. After a period of preliminary training in the army, of which training he did more than he received, he arrived in France at the end of December, 1917, to take up the duties of senior consultant in neuropsychiatry to the American Expeditionary Forces. His work was new and without precedent, as his work had always been. He did it as he had done the work for mental hygiene. The indifference of those over him soon changed to cooperation, and later to enthusiasm, as his own enthusiasm, his ability, his understanding grasp of conditions, his training and his sincerity of purpose began to be recognized by them. His terse manner of expressing his thoughts, the clear logic of his reasoning and the simple charm of his personality, which all who knew him felt, removed many of the obstacles in the path to his goal of efficiency of the army and the proper care of soldiers affected with nervous and mental diseases. It is unnecessary to speak of the details of his organization in France, of the establishment of the neurologic hospitals in the zone of operations, the special base hospitals at La Fauche and Savenay and the psychiatric wards in base hospital groups; these are matters of military history. It is sufficient to say that no department of the medical corps was better organized for efficient service than was the neuropsychiatric. The neuropsychiatric service was Salmon's service, and it was so known; it was his in its conception, and in the working out of its details. It felt his touch and it lived; it lives in the army organization today. The war was the vital thing to Dr. Salmon. His advice was asked, and it was freely given to other departments. He did his bit, whatever it was. I saw him carrying stretchers in France when stretcher bearers were more needed than psychiatrists. What he could do was more important to him than what he was.

Nourished by the white heat of the war, recognition of Dr. Salmon's ability and personality spread. His circle of friends and admirers widened, as more men of varied interests came in contact with him. More men came to know him, and to know him was to love him. He returned from France bearing with him the friendship of many men from many fields, who knew him as his friends among psychiatrists had known him for years. Official recognition took the form of promotions to lieutenant-colonel and colonel in the medical corps, during 1918, and the award of the distinguished service medal. After the war, he was awarded the rank of brigadier general in the Medical Reserve Corps of the army. Personal recognition was accorded in the loyal friendship of all who had worked with him.

When he returned from France in 1919, Dr. Salmon again took up his work with the National Committee for Mental Hygiene until 1921, when he resigned to accept the position of professor of psychiatry in Columbia University, College of Physicians and Surgeons. At the same time, he started private practice in psychiatry. His success as a teacher was due to his singular ability to talk of complex things in a simple manner; no one was in doubt as to what he meant. His simile and metaphor were strikingly apt; his point of view was original and straightforward. He pierced the haze in his subject with the light of a clear thinking mind. His success in practice was to be expected, as for years he had desired to practice and come in contact with patients. His skill at sensing and righting fundamental troubles in his patients appeared uncanny to his colleagues. He had often said that he longed to be a "doctor"; when his opportunity came, he was one of the best, and was happy in his work. University work and private practice brought him in contact with still more people, and resulted in more friends who mourn him.

I have sought to draw a picture of a man, a physician and a friend, using mere facts as a medium. In the face of such facts, praise would be redundant. Thomas W. Salmon lives today in the hearts of his many friends. When the last of them shall have passed, he will live on, for he achieved immortality through the mental hygiene movement, the humane care of the insane and the neuropsychiatric organization of the army, all of which share their immortality with him.

L. C.

VINCENT GILIBERTI, M.D.
1889-1927

Vincent Giliberti was born in Guardia Perticara, Potenza, Italy, on Sept. 28, 1889. He was educated in the public and high schools of New York City and in The College of the City of New York. He received his medical degree at Columbia University College of Physicians and Surgeons in 1913.

Dr. Giliberti was associate professor in the department of neurology at the Post-Graduate Medical School and Hospital and an attending physician in the hospital; also clinical assistant at the Vanderbilt Clinic. He was engaged in the general practice of medicine until five years ago, when he began to devote himself exclusively to patients with nervous and mental diseases.

In the short period of five years, Dr. Giliberti's abilities made it possible for him to assume a prominent place among the neurologists in New York City, and this place was becoming increasingly important. He had an intelligent understanding of the modern conceptions of mental and nervous diseases, and the rapidity and thoroughness with which he familiarized himself with the problems connected with these diseases was unusual. He was a thorough student, and in the short period of five years had trained himself so efficiently that he was able to begin contributing to neurology in a manner that augured well. He made only two contributions, but both show splendid ability efficiently applied. He was co-author of a neurologic study entitled "Postconcussion Neurosis—Traumatic Encephalitis," published in the Archives of Neurology and Psychiatry in August, 1927, page 181. He also wrote an article reviewing the whole subject of injuries of the brachial plexus, which has not yet been published, but which will appear soon.

Dr. Giliberti died on Aug. 6, 1927, of nephritis. He is survived by his wife, Nellie; a brother, who also is a physician, and a sister. Dr. Giliberti was a patient, lovable friend and a scholarly, devoted and dependable associate. A host of friends join in expressing their deep regret at his untimely passing.

News and Comment

PSYCHIATRIC FELLOWSHIPS

The National Committee for Mental Hygiene announces that two fellowships, created by the Commonwealth Fund, are available for training in work in the fields of child guidance, delinquency, education and dependence at the Institute for Child Guidance in New York City. Candidates must: (1) be under 35 years of age, (2) be graduates of class "A" medical schools, and (3) have had at least one year of training in a hospital for mental disease maintaining satisfactory standards of clinical work and instruction. Inquiries and applications should be addressed to Dr. Frankwood E. Williams, Medical Director, The National Committee for Mental Hygiene, Inc., 370 Seventh Avenue, New York City.

COMMITTEE ORGANIZED FOR A MEMORIAL TO DOCTOR SALMON

A committee has been formed to prepare a memorial to Dr. Thomas W. Salmon, professor of psychiatry at Columbia University, and the first medical director of the National Committee for Mental Hygiene. The chairman and treasurer of the committee are Dr. Frankwood E. Williams, medical director of the National Committee for Mental Hygiene, and Dr. Samuel W. Hamilton, assistant medical director, Bloomingdale Hospital, White Plains. The function of the committee is to consider plans proposed for a memorial and to receive funds for this purpose.

INFORMATION REQUESTED ON PSYCHIATRIC CLINICS FOR CHILDREN

The Division on Community Clinics of the National Committee for Mental Hygiene is revising the Directory of Psychiatric Clinics for Children in the United States, issued by the Joint Committee on Methods of Preventing Delinquency in 1925. The cooperation of all psychiatric agencies for the study of children is solicited in order that the directory may be as exhaustive and correct as possible. The division would appreciate receiving the names of all clinics not previously listed, including all clinics organized since Jan. 1, 1925.

NEUROLOGICAL INSTITUTE AT NEW YORK MEDICAL CENTER

Ground was broken on Oct. 19, 1927, for the new Neurological Institute at the New York Medical Center, beside the New York State Psychiatric Institute and Hospital, and a cooperative agreement has been entered into between the new institute and the State of New York for attacking the problems of mental and nervous diseases. The state institution is to be ready for occupany in April, 1928, and the Neurological Institute will be rushed to completion as soon thereafter as possible.

Abstracts from Current Literature

SYMPTOMATOLOGY AND SURGICAL TREATMENT OF TRAUMATIC LESIONS OF THE SPINAL CORD. POUSSEP, Folia Neurop. Eston, **5**:1, 1926.

During the war, Poussep, as director of the Military Neuro-Surgical Hospital at Petrograd, had about 600 patients with spinal injury under his care. Complete records exist of half of them. In 275 cases he practiced surgical intervention. Practically all of the injuries were due to high explosive missiles. A table is given showing the region affected, the nature of the injury and the presence of foreign bodies. None of these lesions were above the third cervical vertebra; the patients injured above this level failed to reach the ambulance. Again, few lesions were found between the third and fourth thoracic vertebrae, probably due to the bony shoulder girdle and to the presence of the heart and great vessels, injury of which would preclude continued existence. Most of the injuries occurred in the lumbosacral portion. In 114 cases, foreign bodies were found in the vertebral canal. According to the author, spinal compressions due to bony fragments or to foreign bodies are of great interest because they give a high percentage of favorable results following surgical intervention. Poussep divides his material logically into complete and partial transverse lesions of the spinal cord, and into cases in which the patients were treated surgically in the early, middle and late stages after their injuries.

Immediately after an acute spinal injury there is complete abolition of tendon reflexes in a large percentage of the cases. Only five out of fifty-nine showed exaggeration. Such abolition does not necessarily signify complete transverse lesions, however, because in some cases the dura was intact and the spinal cord was only severely bruised. The persistence of tendon reflexes, however, indicates a partial or inconsiderable lesion. The abolition of tendon reflexes is explained on the basis of the shock received by the spinal cord. Possibly, also, the contusion gives rise to a large number of punctate hemorrhages, which temporarily bring about this ensemble of symptoms, characterizing complete transverse lesions. Only after from three to ten days, when time is given for the reflexes to reappear, can a more exact diagnosis be made. In a complete transverse lesion of the spinal cord, whatever the level, flaccid paralysis and abolition of tendon reflexes are observed from the beginning and last until death. Moreover, should the lumbosacral region be injured, a degenerating muscular atrophy is to be noted in the lower limbs.

Occasionally this atrophy and muscular degeneration are found associated with edema of the lower portion of the spinal cord. Foreign bodies seem to have interrupted the return circulation in this portion. Moreover, in these cases there was complete failure of muscular excitability in response to the galvanic current. The reaction to the galvanic current returned in one case after the removal of the foreign body. Although the spinal cord was sutured, the reappearance of the muscular excitability evidently was not due to this. Probably the cleaning out of the spinal canal and the extraction of the foreign body permitted reestablishment of the lymphatic and hemic circulation in the lower portion of the spinal cord. The edema of the spinal cord was probably responsible for the loss of electrical muscular reaction. In another case of complete transverse lesion of the spinal cord, in which it was possible

to introduce a probe between the fragments of the spinal cord, suture of the spinal cord was followed in fifteen days by return of tendon reflexes in the lower extremities, and within a month, spasmodic phenomena appeared. In other cases, in which the condition was less pronounced, reappearance of reflexes was noticed after suture of the cord. Such reestablishment never occurred in patients who were not operated on.

It must be concluded from this "that surgical intervention brings about some peculiar modifications in the state of the spinal cord and that debridement and extraction of foreign bodies causes the tendon reflexes to reappear in certain cases. The cutaneous reflexes likewise undergo considerable modification." Cutaneous reflexes usually disappear below the site of the injury in cases of complete transection of the cord. Sensory disturbances are usually pronounced, and in the author's experience all forms of sensibility are deranged at the same level when the lesion is complete. When the lesion is partial, or when there are hemorrhages elsewhere in the spinal cord, the level signs are variable.

After complete lesion, the bladder and rectum are always paralyzed. Retention of urine is the rule; occasionally incontinence of both bladder and rectum is noted. Priapism is rarely observed and is sometimes associated with edema of the penis and buttocks, with or without edema of the lower extremities.

In certain cases, the cardiac pulsations drop to 40 per minute two or three hours after complete section. A few hours later the pulse has quickened, and after from six to seven hours it becomes normal. Usually the pulse quickens and becomes feeble from two to four weeks after the injury. Its acceleration corresponds to a rise in temperature foretelling the end. Dyspnea is present in the initial period in 30 per cent of the cases, even though the inferior thoracic portion is injured. Hiccup also occurs. Abnormal phenomena of the alimentary tract appear the day after the injury in the form of tympanites, which may provoke dyspnea and sometimes vomiting. These phenomena are marked in patients who have edema of the lower extremities. When the upper thoracic region is injured, the symptoms are severe. The symptoms disappear the day after cleansing of the wound and debridement. "The general state of the patient is ordinarily quite good in spite of the severity of the injury. He remains up to the last moment in a state of euphoria which is evidently explained by the fact that he does not feel any pain. This peculiarity has been emphasized by all writers who have observed patients of this type."

In partial lesions of the spinal cord, symptoms differ according to the level, extent and depth of the injury. At the beginning these injuries cause a syndrome of complete transverse lesion, and only rarely at the beginning are the partial lesions recognized. Shock or hemorrhage explains the severe primary manifestations. The symptoms regress more or less rapidly with reappearance of reflexes and of sensation, which always indicate a mild lesion. "Two symptoms of complete transverse lesion have never been observed by me in cases of partial lesion. These are edema of the inferior portion of the body, the abolition of the electric muscular reaction. In the presence of these symptoms, a diagnosis of complete transverse lesion of the spinal cord may be made without hesitation."

The trophic disorders cannot be considered as such unless they come on during the first days and spread rapidly in spite of all measures taken.

After a man has been wounded in the spinal cord, the transportation must be extremely careful, otherwise shock will be still further increased. Poussep had a special train for caring for patients with these spinal injuries. It is of the utmost importance to avoid pressure sores by turning the patient frequently

and by washing the body with a 5 per cent solution of alum. Likewise, retention of urine is to be combatted by the most careful technic in catheterization. The best results were obtained with an indwelling catheter. Retention of fecal material necessitates repeated enemas. Occasionally when two or three vertebrae are damaged, it is necessary to immobilize the spinal column by a plaster jacket.

The question of operative intervention depends to some extent on the time after the injury that the patient is seen, and also on the judgment of the surgeon. Experience has extended the indications for surgical intervention to all traumatic lesions. Poussep divides his material into cases in which the patients were operated on within forty-eight hours after the accident, within the first month, and later, when the wound was old. In the first category, hemorrhage is still active and the clots scarcely formed. In the second category, the hemorrhage has been partially organized. In these cases the dura was usually the seat of scars and adhesions. In the third group dense cicatrices and scar tissue are also found.

Twenty-three wounded men were operated on during the first hours after injury. Eighteen of these appeared to have complete transverse lesions, but in only nine cases was the lesion actually complete. The operation consisted in cutting the skin and subcutaneous tissue down to the spinous processes, separating the periosteum with the muscles down to the transverse processes and then removing the spines and laminae. The spinal canal could then be examined and the blood and foreign bodies removed. If the dura was affected it was opened, and the blood, bone, splinters and foreign bodies were removed. The softened part of the spinal cord was spared as much as possible, but the pultaceous mass in the vicinity was removed by sponging. The wound was then sutured. When the dura was defective the fascia of the adjacent muscles could be used to fill the wound. This was eminently successful, since there was never any escape of cerobrospinal fluid. When the spinal cord was completely separated, Poussep sutured it. Five patients were treated in this manner. Two sutures were passed through the whole thickness of the cord, and these were reinforced by superficial sutures in the pia mater. These superficial sutures alone were insufficient to hold the fragments of cord together. Twelve of the twenty-three patients operated on died. The deaths were unrelated to the type or severity of the lesions; thus, immediate operation is to be considered dangerous because of the open wounds and the spread of infection. This experience led Poussep to postpone operation until the cutaneous wound was healed, the risk of complications being reduced. Seventy-four cases fell into this category; seven of these patients had complete rupture, and in fifteen the cord was crushed. In five of the seven cases the spinal cord was sutured before foreign bodies were extracted. Subsequent to the removal of these foreign bodies spasmodic phenomena and the Babinski sign reappeared in one case. Surgical intervention probably prolonged the life of these persons. The greatest improvement was noted after the extraction of foreign bodies. In cases of incomplete injury, a considerable improvement was often noted, and patients could sometimes return to their work, but, "I have never observed a case of complete reestablishment in patients by partial lesion."

When only compression was present, appropriate surgical intervention gave favorable results, and sometimes these patients were returned to duty.

In certain cases, patients improve spontaneously; among them, operation is either postponed or is not performed. The indications for late operation are aggravation of symptoms, pains, spasmodic phenomena, great weakness or hyperesthesia. Operations become urgent when the upper extremities

are spastic. The aggravation of symptoms is probably due to organized adhesions of the membranes with the spinal canal and subsequent contraction with interference with the hemic and lymphatic circulation. In such cases cysts or dense scarred areas may form. Results were particularly favorable in cases in which such cysts develop. One can follow the progress of symptoms in these cases by the clinical observation pointing to pressure at the periphery of the cord. Before operating it is always well to ask, In what measure is it possible to arrest or suppress the process which is the cause of these serious phenomena? Pain is always an indication for operation, particularly if it is radicular in type.

On examination the cerebrospinal fluid sometimes gave indications for or against intervention. Xanthochromia and a high albumin content, with or without pleocytosis, indicated operation.

Conditions found during operation in these late cases were cysts, scars, sclerotic areas, and foreign bodies. Cysts filled with clear or yellowish fluid were found in nearly one half of the cases. The spinal cord was sometimes thin and firm, and the vessels of the cord were interrupted at and below the area of strangulation. The adhesions to the spinal cord were sometimes of extreme density, giving rise to the so-called traumatic circumscribed spinal meningitis. These patients suffered from interference with the circulation, and myelolysis was indicated for the freeing of the spinal cord from these dense cicatrices.

In cases in which the roots presented serious damage with deformity, the cicatricial region could be resected and the ends sutured. In eight cases of this type, with lesions including from four to eight nerves, use of the limbs and even walking was possible after from six to eight months; anesthesia had practically disappeared. Some of these patients were operated on eighteen months after injury, but still the nerves of the cauda equina continued to react to the electric current. Sometimes such intraspinal radiculolysis was impossible and resection of the whole cicatrix would have been necessary, but this would have been of questionable value. The operation of radiculolysis consisted of separating the nerve roots as well as possible and removing the foreign bodies. On the whole, injuries of the cauda equina were followed by a most satisfactory surgical result.

In certain cases in which favorable results are not obtained by myelosis, a partial reestablishment of the conductivity of the cord by a sidetrack has been obtained. The injured part of the spinal cord and the parts above and below are uncovered, the anterior and posterior roots above and below the lesion are then exposed and cut as close as possible to the point of entry into the spinal canal. These ends are then sutured. There was diminution of the spasmodic phenomena four months after operation in all such cases. The anesthesia of the lower limbs was reduced and the deep sensibility was brought back. The patients could walk with crutches. "I explain this improvement by the fact that due to the reunion of roots there is produced a development of descending collateral ramifications which exist in all the spinocerebral tracts and which take on in part a new function." A few operations on the roots and for injuries due to stab wounds and fractures of the vertebral column are given.

In discussing his results, the author states that surgical intervention benefits most of the patients, and that in certain cases this amelioration is considerable. Early operations are dangerous, resulting in 50 per cent mortality, due to infection. Aside from these the mortality has been 3.5 per cent and in late operations only from 1 to 1.5 per cent. The opening of the dura does not notably increase the danger of the operation. Death has always been due to

infection and never to the escape of cerebrospinal fluid. Suture of the spinal cord after complete transverse lesion has given no perceptible return of function. When the section is incomplete, however, suture brings about amelioration of all the symptoms. Extraction of foreign bodies is perhaps the most helpful procedure. Late operations on the cord have given the best results, owing not only to the removal of foreign bodies and radiculolysis, but also to the possibility of suturing the roots. Operations of sidetracking have given satisfactory results, but new studies will be necessary.

WALTER FREEMAN, Washington, D. C.

SUBARACHNOID HEMORRHAGE. E. HERMANN, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:667 (Nov.) 1926.

Hermann reports twenty-four cases of spontaneous subarachnoid hemorrhage from the clinic of Flatau in Warsaw. The subject has only recently been separated into a clear clinical entity. The work of Eskuchen appeared in 1919, followed in 1923 and 1924 by the reports of Goldflam, Matzdorff and Meylan. Previous to this there had been scattered reports of cases and brief textbook descriptions. Serrés (1819) was the first to differentiate meningeal from cerebral hemorrhages. In 1857, Baillarger pointed out that the subarachnoid space was a frequent seat of hemorrage. In 1859, Wilkes reported a case of spontaneous subarachnoid hemorrhage with necropsy, and in 1883, reported three clinical cases. Guitrac (1869) collected thirty-four cases, two of his own, and reported them in his textbook. Hayem (1872) reported four cases, and in 1904, Froin reported on the cerebrospinal fluid in subarachnoid hemorrhage. Ehrenberg (1912) gathered thirty-one cases; later, Fearnsides gathered forty-four cases, and more recently Symmonds reported on 127 cases of spontaneous subarachnoid hemorrhage which he had gleaned from the literature. In the Polish literature, Flatau was the first to give a good description of subarachnoid and subdural hemorrhages. In the German literature, descriptions of this entity are recent. Mention of it is not made in the 1923 edition of Oppenheim's textbook.

The question of terminology arises. Shall these cases be spoken of as leptomeningitis haemorrhagica, or as hemorrhagia subarachnoidalis? Hermann believes the former term should be reserved for cases secondary to inflammation of the meninges, as in hemorrhagic epidemic encephalitis, syphilitic meningitis, epidemic meningitis and other conditions. The term hemorrhagia subarachnoidalis should be used for those cases of spontaneous subarachnoid hemorrhage resulting from aneurysms, vascular lesions, hemorrhagic diatheses, blood diseases and hypertension. Flatau and Goldflam favor the term subarachnoid hemorrhages, but Hermann considers this too general. Further, there is no unanimity as to what is to be considered a spontaneous hemorrhage. Matzdorff considers those cases in which there is a known etiology as cases of spontaneous subarachnoid hemorrhage. Hermann, on the other hand, considers those cases of subarachnoid hemorrhage for which no etiology is clear as cases of spontaneous hemorrhage, and calls them essential subarachnoid hemorrhages. Goldflam also designates those cases of subarachnoid hemorrhage for which no extraneous cause can be shown as spontaneous hemorrhage.

The etiology of subarachnoid hemorrhage is varied. The causes may be grouped as follows: (1) Arteriosclerosis—hemorrhage occurs in this condition as the result of a degenerative sclerosis of the pial vessels (Dejerine, Richon et al.). (2) Aneurysms of the base of the brain (Oppenheim, Symmonds, Ingvar, Widal et al.) or in the spinal canal (Etienne, Robow). (3) Syphilis of the

vessels. This may be of three types: degenerative, causing aneurysm; inflammatory, with inflammation of the meninges, and an endarteritis of typical type, syphilitic in origin. Hermann had two cases in which the subarachnoid hemorrhage was of syphilitic origin. One occurred in a man of 26, with a positive fluid and a negative blood Wassermann reaction, and the other was in a woman of 37 with petit mal, left hemiplegia and aphasia, who showed a syphilitic endarteritis of the vessels at autopsy. (4) Miscellaneous causes which may result in a transitory hypertension, as in epilepsy (Gowers), tetany, tetanus, uremia (Chaufford, Roger), strychnine poisoning, chorea, pertussis and at birth. The relation of the epilepsy to the subarachnoid hemorrhage is still in question. Some hold that the epilepsy is secondary to the hemorrhage, and occurs as a result of irritation of the cortex. Mackiewicz reports a case of subarachnoid hemorrhage which was first indicated by jacksonian attacks. Others believe the epileptic attack is primary and the subarachnoid hemorrhage secondary. Hermann reports two cases of subarachnoid hemorrage with histories of epilepsy, in which the hemorrhage occurred independently of an attack. (5) Cerebral trauma or contusion. (6) Intoxications such as lead, alcohol, nicotine and carbon monoxide. (7) Meningitis of varied origin; cerebrospinal meningitis, tuberculous meningitis, pneumococcic and syphilitic meningitis; also meningitides of unknown origin. Apert observed subarachnoid hemorrhage in a child, aged 4½, as the first symptom of abdominal typhoid fever. It occurs also in typhoid recurrens, Malta fever, diphtheria, measles, smallpox, influenza, pneumonia, acute nephritis, sepsis, acute rheumatic fever, malaria, epidemic encephalitis, angina, rubeola, tuberculosis, thrombophlebitis, and in diseases of metabolism such as gout and cholemia. Cases have been reported in all the above conditions, and almost any infectious fever may cause subarachnoid hemorrhage. (8) Hemorrhagic diatheses such as leukemia, scurvy, pernicious anemia and hemophilia. (9) Cerebral and spinal cord tumors, especially those involving the meninges, may cause small subarachnoid hemorrhages.

Despite this imposing array of causes of subarachnoid hemorrhage, there is a group for which definite etiology can not be found. Symmonds collected forty-one of these cases from the literature. These are the true cases of spontaneous subarachnoid hemorrhage; even examination at necropsy fails to indicate a cause for the hemorrhage. One is forced to fall back on the concept of Cohnheim that in these cases there occurs a diapedesis of blood cells as a result of dilatation of the cerebral vessels and slowing of the blood flow. Goldflam believes in a nervous origin of these hemorrhages, and spoke of "angioneurotic hemorrhages"; he called attention to the fact that migraine plays a not unimportant rôle in them. Blood escapes into the subarachnoid space in these cases by rhexis and by diapedesis. In cases of degenerated vessels, rupture occurs and the hemorrhage occurs by rhexis. If the hemorrhages occur in inflammatory states, there occurs vasodilatation, slowing of the blood stream, and an escape of blood by diapedesis.

Most of the patients in Hermann's series of twenty-four were under 35. Of these, ten were men and fourteen were women. The etiologic factors were: arteriosclerosis, 2; syphilis, 2; trauma, 2; cardiac failure, 2; nephritis, 1, and epilepsy, 3. In twelve cases etiologic factors were not demonstrable. These were the cases of idiopathic subarachnoid hemorrhage which occurred in young persons. Goldflam said most of the cases occur in the fall and winter; Hermann found his cases rather evenly distributed over all the seasons.

The onset of subarachnoid hemorrhage is sudden, typically with sudden severe pain in the neck and the back of the head. Unconsciousness of varied depth and duration follows shortly after. There is usually marked activity

and excitement. The loss of consciousness may last from a few hours to ten days — often the onset is with lethargy which becomes deeper and continues until death. Delirium and dementia are observed sometimes. Psychic disturbances, such as Korssakow's syndrome, have been reported in cases by Flatau and Goldflam. The severe headaches are usually accompanied by vomiting. The latter may persist for a few days or last only a short time. Symptoms of meningeal irritation such as stiff neck, Kernig and Brudzinski signs, and slowing of the pulse are seen. The temperature is subnormal at first, but later becomes elevated, rarely over 39 C. The temperature lasts a few days and then drops to normal. Various signs of collection of blood on the cortex may be seen: jacksonian attacks, hypertonia generalis, hemiparesis, aphasia, conjugate deviation and other symptoms. In only six of the twenty-four cases reported by Hermann was a hemiparesis demonstrable.

Hermann says that the onset need not be sudden. There may be prodromal symptoms for some time before — vertigo, headache, tinnitus and sleep disturbances. Certain signs may develop during the disease: changes in the pupils or the ocular fundi; choking has been seen, but is not common; hemorrhages in the retina occur and are simultaneous with edema and hemorrhage in the sheath of the optic nerve — it occurred in three of Hermann's cases. Hermann stresses an important point to the effect that in cases of subarachnoid hemorrhage, there are apt to be small hemorrhages also within the substance of the brain. The spinal fluid is bloody, and the spinal fluid pressure is increased, xanthochromia occurs after the hemorrhage is old. A pleocytosis need not mean meningitis, because it may occur as the result of irritation to the meninges by the red blood cells.

ALPERS, Philadelphia.

DIRECT AND REFLEX PARALYSES IN EXTRAPYRAMIDAL LESIONS. MAX EGGER, Schweiz. Arch. f. Neurol. u. Psychiat. **19**:39, 1926.

The older anatomists had taught that skeletal muscles possess two insertions, a superior and an inferior; if the muscle from its superior insertion pulled on the inferior one, its contraction was called centripetal; if, on the contrary, the contraction pulled on the superior insertion, it was called centrifugal. In more recent times, this conception is not considered of importance, since the pull may take place in either direction, according to whether the insertion is fixed or free. The author, however, considers the terms "centripetal" and "centrifugal" as used in reference to the direction of pull of a given muscle as being of rather profound biologic significance. Electric excitation of the motor cortex produces only centripetal contraction such that flexion, extension, pronation, supination, abduction and adduction are of the same type as the ordinary voluntary contraction. Excitation, however, of the cerebellar nuclei produces only tonic contractions of the distant muscles combining to produce a fixation of the limb in a static consolidation. This contraction, while not actually centrifugal, resembles it closely. In the case of movement of the tibia in the condyle of the femur, certain proprioceptive stimuli arise which produce engrams in the nerve centers, which are essentially different from those produced by the sensation of the movement of the tibia on the femur. It might be assumed that there is a special neuromuscular mechanism corresponding to these centripetal and centrifugal contractions. From the standpoint of clinical observation, there seems to be a different mechanism for muscular movement, depending on whether the muscles are used for free voluntary movements or for the purpose of locomotion. In cases of capsular hemorrhage

with hemiplegia it is possible for a person to walk and to use certain muscles in this function, which when tested, either in a sitting or lying position, seem paralyzed. It is possible that this conservation of the function of walking is controlled by the extrapyramidal system, while the power of volitional movement controlled by the pyramidal system may be completely abolished. The author further suggests that in locomotion the contraction of the muscles is centrifugal, while in volitional movement it is centripetal. Another example which he quotes is that in a given case of anterior poliomyelitis the semimembranous and semitendinous muscles may be paralyzed so that they are unable to flex the leg on the thigh, but still may be able to produce extension of the pelvis. The study undertaken was the attempt to prove the principle that a terminal motor organ such as a muscle can be paralyzed in one of its functions but retain another, and that the centrifugal contraction of the muscles may be lost with preservation of the centripetal movement, and vice versa. The discussion also deals with the nervous mechanisms concerned in these functions. The case of a patient who had had a vascular lesion, of syphilitic origin, in the region of the midbrain and red nucleus was demonstrated to support some of the theories. This patient had a paralysis of the right third, fourth, fifth, sixth and seventh cranial nerves, with a superficial and deep hemianesthesia on the left side. Volitional motor power was conserved, but on the left side there was marked hemiataxia with choreo-athetotic movements and marked cerebellar asynergia. The patient was able to walk only with two other people supporting her arms. The left lower extremity was held in a position of hyperextension, and while she made powerful contractions of all muscles, she was totally unable to walk alone or to sustain her weight on the left lower extremity. Moreover, in walking, as soon as the knee became flexed to the least degree, the whole limb gave way, and suddenly she fell heavily to the ground. She was totally unable to raise the foot off the ground, and in climbing stairs dragged the limb helplessly after her. The surprising feature, however, was that when the strength of the extremities was tested while she was sitting or lying, contractions were powerful and practically equal on the two sides. From this case the author assumed that it was possible to have complete conservation of volitional power, as governed by the pyramidal system, but at the same time marked dysfunction of the neuromuscular apparatus governed by the rubrospinal system.

Friedreich's ataxia is a disease in which some of the foregoing phenomena have also been noted. Seldom is there any direct paralysis of the limbs; the difficulty in locomotion seems to be due to the ataxia, which is the outstanding feature of the disease. It is known that in this condition the disease shows a predilection for the cerebellar system, above all for the afferent tracts, such as those of Gowers and Flechsig. The author studied fourteen cases of the disease to estimate whether there was a disturbance of muscular action corresponding to that already described. In spite of the Babinski sign, indicating an involvement of the pyramidal tract, there was no disturbance of voluntary power, which was in strong contrast to the difficulties the patient had in attempting to use the limbs for locomotion. In testing the function of the upper extremities, the patient lay face downward on the ground, and was instructed to raise the body by the arms. The idea was that in this situation contraction of the muscle would be centripetal rather than centrifugal. In like manner, when the lower extremities as well as the ordinary functions of walking were tested, the patient was made to arise from a kneeling position, to jump with the feet approximated, and to jump from a chair to the ground. In all these cases, while the muscular strength as tested by the dynamometer

either in the sitting or lying position was fairly good, obvious paralysis was not demonstrable. Muscular movements under the conditions of walking, standing and in the tests already mentioned were remarkably deficient. This was considered by the author to indicate that there was a neuromuscular mechanism in which the contractions of the muscles were centrifugal, and that it might be interfered with by disease of the rubrospinal or the afferent spinal cerebellar system.

PARKER, Rochester, Minn.

THE PATHOLOGIC ANATOMY AND PATHOGENESIS OF ACUTE ASCENDING PARALYSIS (LANDRY). L. I. SMIRNOW, Arch. f. Psychiat. u. Nervenk. **78**:585 (Oct.) 1926.

The author reports two cases of acute ascending paralysis and discusses the syndrome in general. The first case is that of a man, aged 24, who a short while after an attack of diarrhea, during a course of treatment with quinine for malaria, developed a flaccid paralysis of both lower extremities; in a few days this spread to the upper extremities and bulbar centers. Disturbances of sensibility did not occur. Seven days after the onset, the patient died. Postmortem examination showed infiltration, with hyperemia, thrombosis and extravasations over the whole central nervous system from the cerebral cortex down to and including the spinal roots. There was marked dilatation of the blood vessels with widening and infiltration of the periadventitial spaces. In the parenchyma of the central nervous system, the pathologic process was predominantly localized in the gray matter. There was moderate hyperplasia of the neuroglia; in the cortex this hyperplasia, with an accompanying neuronophagia, was marked. The ganglion cells showed acute reaction, mostly in the form of swelling. There was diffuse dismyelinization in the cord but none in the brain. The central canal showed marked proliferative phenomena. The white substance was affected much less and only near the more intense foci in the gray substance (this was most marked in the lower cervical and thoracic segments of the cord). The intensity of the process in the cord showed gradual diminution from the second lumbar downward, and there the disturbances seemed to be restricted to the central canal and adjacent structures. The author regards the symptoms as manifestations of an acute toxic meningopolioencephalomyelitis and radiculitis. He believes that the acute diarrhea must have been due to the same toxic agent. The changes in the central nervous system did not suggest the typical nerve complications of malaria.

The second case was that of a man, aged 28. The onset of the disease occurred, one month before death, with fever and pains and paresis in the lower extremities. These spread gradually upward to the upper extremities, and then involved the bulbar motor nuclei. There were no sensory disturbances. Albumin and casts were present in the urine shortly before death. The postmortem observations, in contradistinction to those in the first case, were mostly of the degenerative type, without infiltration and with only slight proliferation. The cerebrum did not show any involvement. In the cerebellum there was moderate diffuse involvement of the Purkinje cells. There was acute swelling of the ganglion cells in the medulla oblongata apparently of rather short duration (probably terminal symptoms). The maximal changes in the cord were in the lumbar enlargement with diminution in severity both upward and downward. The reaction here was mainly characterized by degeneration of the ganglion cells of the anterior horns and of Clarke's column. The neuroglial reaction was rather weak and reached its highest degree in the zonal layer of the lumbar enlargement. There was diffuse dismyelinization in the whole

periphery of the cord which, however, showed no tendencies toward system degeneration. All tracts, descending and ascending, were equally involved. Here and there were isolated foci, characterized by dropping out of cells and nerve elements in general, without the least attempt toward repair or scar tissue. The only sign of activity was seen in the ependyma of the central canal, where there was a marked proliferation of the ependymal cells and a marked pericanalicular ependymal infiltration. In addition to these changes in the central nervous system there was an acute glomerular nephritis. In neither case were anatomic changes found in the peripheral nervous system.

The author comes to the following conclusions: 1. Anatomically there seem to be five groups of Landry's paralysis: (*a*) with no appreciable microscopic changes in either the peripheral or the central nervous system; (*b*) with changes in the peripheral nervous system only; (*c*) with changes in the spinal cord only; (*d*) with changes in both the spinal cord and the peripheral nervous system; (*e*) with involvement of the whole cerebrospinal axis (as in the two cases reported).

2. There is no one pathogenic factor, either bacterial or toxic, that can be regarded as the specific causative agent of Landry's paralysis. In the literature there seemed to be as many causative agents as there were cases reported.

3. The clinical picture, although usually that of a more or less acutely progressing ascending paralysis without sensibility disturbances, shows, nevertheless, much variation both in the duration and different manifestations of the disease process.

4. As can be seen from the two cases reported, as well as from other cases in the literature, the peculiarity of this disease process depends on either one or the combination of two factors: hypertoxicity of the causative agent, and marked decrease of the resistance of the central nervous system as compared with other organs. From either of these, or from a combination of them, the central nervous system breaks down without appreciable resistance to the invading agent. In the first case there were marked proliferative and infiltrative reactions, indicative of the malignancy of the invading agent, without any attempt at regeneration; in the second case, in which the virus was probably more benign, there seemed to be only a degenerative reaction of an apparently nonresistant central nervous system.

5. The central canal in both cases seemed to play an important rôle as a portal of entrance into the central nervous system. It was not the sole one, however, for there were signs suggesting that all five possible modes of entrance were utilized: blood vessels, lymph vessels, central canal, contiguity and the subpial spaces.

MALAMUD, Foxborough, Mass.

THE NEUROTROPIC VIRUS. F. BREINL, Arch. Path. & Lab. Med. **2**:386 (Sept.) 1926.

The author defines the term "neurotropic virus" as meaning all those invisible and filtrable viruses which show special affinity for the central nervous system and being inoculated into the organism, proceed along the nerves to the central organ where they undergo their strongest multiplication and produce lesions that are deleterious to the whole organism. As the tropism is not limited to the nervous system in all instances but may affect also the epithelial layers of the skin, the cornea and the mucous membranes, the more general term "ectodermotropic" would be more correct. Levaditi has designated the diseases — poliomyelitis, rabies, vaccinemia, herpes, herpes zoster, and chickenpox, produced by this group of viruses—the neurotropic ectodermoses.

All the neurotropic viruses have the common characteristics of invisibility, ability to pass through filter candles and to survive for a long time in glycerol as well as in the dry state; of being uncultivatable artificially, of the production of similar anatomic lesions in the nervous system, and of neutralization of the homologous virus, in moderate amounts, by the serum of a convalescent patient. In spite of their similarities each of the group represents an absolutely specific antigen. They are related to each other systematically but not genetically.

Breinl discusses herpes, herpes zoster, vaccinia, poliomyelitis and rabies at length. From twenty-four to thirty-six hours after the inoculation of the contents of an herpetic vesicle into a rabbit's cornea, minute vesicles are formed at the point of inoculation and marked desquamation of epithelial cells occurs. These epithelial cells are edematous, show vacuolar degeneration of the cytoplasm, degeneration of the chromatin structure of the nucleus and formation of intranuclear, acidophilic, small, round bodies. Lipschutz has described minute granules within these bodies, which he considers the specific agent of the disease. Others, however, though they admit these granules are of diagnostic importance, think they are only fragments of the disintegrated nucleus.

The virus propogates from the eye to the brain along the nerves, proceeding in the axis cylinders. The neurologic manifestations correspond to the part of the central nervous system affected by the virus — after intracerebral inoculation, in the meninges, after ocular infection in the mesencephalon and medulla. The ganglion cells show nuclear swelling and the nuclear changes resemble those occurring in the epithelial cells. The changes in the brain begin with hyperemia, edema and hemorrhages, intrameningeal and intracortical. This is followed by cellular infiltration with lymphocytes, leukocytes, plasma cells and macrophages, located either perivascularly or in independent foci. If the animals survive long enough, the cells of the exudate disappear and are replaced by glia. The pathologic picture is similar to that of human encephalitis.

The herpes virus is found in man in the lesions, in the saliva, and sometimes in the cerebrospinal fluid; in the last two instances, even when the person was lesion-free. It is not known whether it is the same as the virus of encephalitis or not. Infection of the cornea of a rabbit is followed by a period of immunity of about six months' duration and the successful reinoculations after this time show a milder course.

Herpes zoster seems to be a different entity from herpes simplex. Lipschutz describes inclusion bodies in the nuclei of the epithelial cells which, in his opinion, are related to the virus. His observations on herpes zoster and chickenpox lend support to Bokay's theory of the identity of the two diseases.

Vaccine virus, like that of herpes, shows a special affinity for the skin, cornea and brain, but its affinity for the nervous system is considerably less than that of herpes. Infection with vaccinia produces immunity of the whole body and this immunity can be transferred from an immune animal to a normal one by the transplantation of a patch of skin.

The virus of poliomyelitis and rabies multiplies exclusively in the nervous system. That of poliomyelitis proceeds from the periphery to the central nervous system along the nerves, probably in the nervous substance as well as in the perineural lymphatic vessels. The greatest multiplication of the virus takes place in the gray matter of the spinal cord. There the lesions consist of edema, hemorrhages and cellular infiltrations, partly perivascular, partly as independent foci. This infiltration is first of polymorphonuclear leukocytes, later of mononuclear and fixed tissue cells. The ganglion cells show

swelling, formation of vacuoles and fading of the Nissl bodies. During recovery the focal infiltrations and necroses are replaced by scar tissue. Changes in the other organs are not distinct. According to Flexner and Noguchi, the virus can be cultivated, under anaerobic conditions, in the form of small corpuscles. Although no virus is found in the blood, spinal fluid or parenchymatous organs, it is present in the mucous membranes of the nose and throat, where it may persist for six months. Immunity in man is of limited duration, and the serum of the convalescent man or monkey neutralizes the virus in vitro. This may be of diagnostic value in doubtful cases. Flexner and Lewis have prepared a strain of the virus which immunized monkeys after a mild course of the disease, and this serum has prophylactic properties if administered intraspinally early enough.

The characteristics and manner of propagation of the rabies virus closely resemble those of poliomyelitis. It is possible to infect intracerebrally, subcutaneously, intracorneally and intracutaneously, without, in the latter instances, any noticeable symptoms at the point of inoculation. The virus may remain latent in the brain and the disease break out later following nonspecific insults such as trauma, cold and so forth.

PEARSON, Philadelphia.

A NEW TYPE OF HEREDODEGENERATIVE DISEASE LOCALIZED IN THE STRIATE BODY AND CORTEX WITH EXTENSIVE MYELOLYSIS: ITS DISTINCTION FROM THE PSEUDOSCLEROSES. B. OSTERTAG, Arch. f. Psychiat. u. Neurol. **77**:3 (June) 1926.

The author presents the clinical and pathologic pictures of a case of what he considers to be a special type of heredodegeneration. The patient's mother had an attack of chorea with slight psychic disturbances at the age of 21 (four years after the birth of patient), following rheumatism with endocarditis. She recovered from this attack and had no recurrence of the chorea. The patient had an attack of rheumatism with endocarditis at the age of 11 (1916), and following this, an icterus that was considered infectious. Slight restlessness in movements was noticed even then. About a year later, definite motility disturbances and tremors were noticed, and then disturbances of swallowing and tremors of the right arm and leg developed. As time went on, disturbance of speech, increased salivation, stiffness of facial expression and some psychic disturbances developed. In 1919 (three years after onset), she could not attend school because of inability to control her movements. Psychically, there was some irritability and retardation. In 1920, she was admitted to the hospital.

When examined she showed: expressionless facies; uncontrollable emotionalism; involuntary movements of the head, trunk and other musculature on attempted voluntary motorization and coarse intention tremor. Later in 1920, there appeared athetoid movements, psychic irritability, beginning flexion contracture in the left leg, an achilles clonus, and further difficulty in swallowing. The spasticity increased gradually. The right leg began to show contractures also. Main-en-griffe developed in both hands. Twitching in the legs and arms, and, later, in the facial musculature developed, and following this, signs of definite pyramidal tract disease with a Babinski sign on both sides. With that there was gradual mental deterioration, marasmus, and sepsis following decubitus with death five years after the onset of rheumatism.

The postmortem examination showed, among other things, cirrhosis of the liver with fatty degeneration and necrosis of parts of the parenchyma, with

an increase of connective tissue and the beginning formation of parenchyma insulae. The brain showed marked internal and external hydrocephalus and, even superficially, a visible sinking of the tissues over the frontoparietal regions on both sides. On section, there was marked atrophy and softening of the caudate nucleus, a symmetrical softening extending from the ganglia up into the frontal and parietal regions, and, in places, even into the temporal region.

Microscopically, the whole neostriatum showed marked shrinking with processes of softening in the caudal parts. The marked shrinking in the head of the caudate nucleus made it appear as if the cellular elements were increased. Neostriate cells were all affected, but the smaller cells much more so than the large ones. There was a proliferation of the plasmatic neuroglia and, in the putamen, an attempt here and there at fiber formation. A similar process was seen in the extension into the cerebral cortex where the destruction was even more marked, with marked status spongiosus and mesenchymal proliferation. There was rather scanty glia proliferation. Some of the glia cells resembled the Alzheimer type, but were not identical with it. There were other atypical neuroglia cells, and, in the striate as well as cortex, a great many gitter cells and fat droplets in the adventitial spaces. The striothalamic and striopallidal tracts stained poorly. Disease of less extent was noticed also in the pallidum and corpus luysii, with shrinking, destruction of cells and loss of nerve fibers. The substantia nigra and red nucleus were affected to a much less extent, whereas the dentate nucleus seemed to be fairly well preserved. The white matter of the cerebral hemispheres showed diffuse softenings in different regions. At the confluence of several small areas of softening to form one large area, one could still see some more or less intact fibers, and the fields of destruction showed many gitter cells with much fat. The softening took the form of a myelolytic process, there being no signs pointing toward the vessels as causative factors, certainly no emboli.

In discussing the case and in the attempt to establish its relation to the Wilson-pseudosclerosis group (heredodegenerative), as well as to show its distinction as a special type, the author emphasizes the following facts: The similarity of tendencies in mother and daughter, the development of extrapyramidal disease following an infection which in the patient manifested itself first as a hyperkinetic syndrome followed later by extrapyramidal rigidity and other cerebral signs, especially pyramidal symptoms. The pyramidal symptoms, however, reached a degree which is not commonly encountered in Wilson's disease and can only be explained on the basis of the extraordinarily extensive lesion which, toward the end, gradually encroached on the pyramidal tracts.

As to the pathologic conditions, the author stresses the extensive myelolysis as the most characteristic of this type and the one that distinguishes it from the hitherto described heredodegenerative diseases. In discussing the pathogenesis, the author sees in the exogenous factor a precipitating cause only, which acted on a constitutional inferiority in the central nervous system. This inferiority showed itself in the special relation between diseases of the hepatic and central nervous system, in the peculiar myelolytic tendencies, in the special type of reaction of the parenchyma of the brain which is characterized by greater vulnerability of certain grisea (neostriatum), and the peculiar neuroglia reaction which shows an insufficiency in scar formation as well as production of atypical Alzheimer-like cells.

These factors, anatomic as well as clinical, are, according to the author, sufficient criteria for the establishment of a new disease entity.

MALAMUD, Foxborough, Mass.

PATHOLOGICANATOMIC CHANGES IN THE CHRONIC FORMS OF EPIDEMIC ENCEPHALITIS. L. OMOROKOW, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:421 (Sept.) 1926.

Economo first promulgated the view that the pathologic process in epidemic encephalitis is lymphogenous, and that the virus gains entrance from the subarachnoid spaces. It has a particular affinity for the gray matter. Economo felt that the vascular changes, viz., the perivascular infiltrations, were secondary because they occurred only in the gray substance. Others have differed from this view. Globus found perivascular infiltration in the acute forms of encephalitis, while in the chronic forms he found progressive intimal and adventitial changes, as well as gliosis and secondary parenchymatous changes. He divides the subacute forms in a latter paper with Strauss into three types: (1) acute infiltrative forms; (2) acute hemorrhagic forms; (3) subacute reproductive form. Klarfeld and Herxheimer, as well as many Russian pathologists—Kulkow, Kononowa, Emdin and others agree with this view. Zeko recognizes three types of changes in the chronic form: (1) degenerative changes in the parenchyma of the brain with glia proliferation; (2) slight inflammatory changes in the vessel walls; (3) marked edema of the pericellular and perivascular spaces. The process involves all the gray masses of the brain stem and the nuclei of the cerebellum, while the inflammatory changes are most marked in the vessels of the substantia nigra, subthalamic area, red nucleus and pons. Luksch has described peculiar nuclear inclusions in the ganglion cells, which Mittasch later found also in the cytoplasm. Herzog found them also in the floor of the fourth ventricle.

According to the recent work of Levaditi, Kling, Talmier, Piazza and others, encephalitis is caused by a specific virus which passes through a Mandler filter. Talmier obtained cultures of the virus in Noguchi's medium. In thirty-five cases he obtained the virus from the spinal fluid, in seven cases from the brain and in five cases from the secretion of the larynx. He injected the virus subdurally into 295 rabbits; 167 died after from two to six weeks, and in 108 of these definite changes in the brain were found from which the virus could be extracted. This is in contradistinction to the work of Rosenow on a streptococcus as the cause of encephalitis, and also to that of Kulkow on the rôle of a diplococcus in encephalitis.

Omorokow describes the pathologic changes in two cases of chronic encephalitis, both of which showed typical pictures of parkinsonism. The meninges showed nothing significant save round cell elements of an unspecific type, such as Metchinkoff found in serous meningitis, Diamond in tuberculous leptomeningitis, and Fulci in anthrax leptomeningitis. These cells arise from the vascular endothelium and are phagocytic. The vessels in the cortex showed no specific changes, and were of two types, an acute type with dilatation of the perivascular spaces, venous and arterial hyperemia, as well as changes in the endothelium which were characterized by swelling of the nuclei and separation of the endothelium from the vessel walls. The chronic changes consisted of hyaline degeneration of the vessel walls. The cell changes consisted of the acute cell disease of Nissl. They were most prominent in the cells of the cortex and in the subcortical ganglia. The nuclear inclusions described by Luksch were found by Omorokow. In the nuclei of pigmented cells in encephalitis, Luksch found small round or oval inclusions which occurred singly or in numbers. They are smaller than a nucleolus, homogeneous, sharply defined and take a brown or yellowish stain. They are similar to the bodies found by Joest in bornas, a lethargic disease of horses. Luksch found them also in a control

case of nonepidemic encephalitis, in the pigmented cells of the substantia nigra. Omorokow found them chiefly in the "subcortical ganglia" and in the cortical cells of the uppermost layers in the precentral convolution. The ependymal cells of the third ventricle as well as the cells of the choroid plexus showed definite changes. The ependymal cells of the third ventricle showed a loss of their cubical contour, with deformity of the nucleus. They contained small bodies, about the size of a glia nucleus, as well as layers of pigment. The cells of the choroid plexus also contained much pigment.

Omorokow calls attention to the fact that in these cases of chronic encephalitis the chief changes were in the ganglion cells of the cortex and in the changes which were found in the ependymal cells and the choroid plexus in the region of the third ventricle, particularly in the region of the thalamus. The occurrence of ependymitis granularis is well known in general paralysis, in cysticercus of the brain and in hydrocephalus. Binswanger considers it a characteristic condition in general paralysis, but admits that it occurs in other diseases. Weigert, on the other hand, considers it a normal process, and Brodmann failed to find it only once in eighty brains; this was a young subject. Others disagree with this view. Beadles asserts that no ependymal granulations occur in normal persons, while Perando found them only sixty-five times in 140 cases. Saltykow examined 120 brains of normal men and found a more or less marked ependymitis in all. Omorokow calls attention to the work of Jeremias who observed in the early stages of ependymitis an enlargement of the roof epithelium which followed overgrowth of neuroglia. He considers the observations in this case to be similar to this. He agrees moreover, in view of the changes which he found in the cells of the choroid plexus, with the view of Economo: that the intoxication in encephalitis comes through the cerebrospinal fluid and not through the blood vessels. He thinks it possible that the infection comes from the pharynx, is carried through the lymph system to the cerebrospinal fluid, and from there to the central nervous system, particularly to the ependyma of the third ventricle and the choroid plexus, and to the hypothalamus and cortex.

ALPERS, Philadelphia.

NEUROTIC CHARACTER. E. GLOVER, Brit. J. M. Psychol. **5**:279, 1926.

Certain personalities which are treated by the psychiatrist cannot be classified in the usual categories. Their symptoms are rather vague and usually include diffuse hypochondriacal preoccupations, mild fears, and vague suspicions. Two sets of facts stand out: (1) the individual is faced with recurrent crises of a stereotyped form; (2) these are associated with some change in the "libidinal milieu." Two stock situations recur with monotonous frequency—one in which the individual has a series of reverses or a "run of hard luck" and the other in which he unwittingly and through good intention brings unhappiness to certain significant persons in his environment. He comes to be known among his friends as "his own worst enemy" or a "pest." As an example of the activity of such an individual, one sees the man who, in trying to amass a fortune, continually makes investments which end disastrously. He seems to be unable to profit by experience. Another example is found in the individual who shows considerable skill in building up a business, and then has disaster tear down his achievements, usually because of an avoidable mistake. There is also the individual who is constantly shifting from one occupation to another, such as an artist who becomes an actor, then a teacher, and finally has a nervous breakdown. Other persons drive themselves in a compulsive way into one love entanglement after another

or into some other complex emotional situation. The situations are repeated in a very stereotyped way and disaster in one form or another seems to be the inevitable outcome.

Such individuals have been designated as "neurotic characters" and have three features in common: (1) the reaction is pathologic; (2) it is diffused throughout every-day life; (3) it is supported by rigid and frequently convincing rationalizations. In these cases the whole personality is permeated with reactions which, if concentrated, would bear a striking similarity to a neurotic symptom, which is a localized expression of the same process that leads to a sweeping involvement in the neurotic character.

Psychoanalysts have made many contributions to the study of character traits. The classification of orderliness, obstinacy and avarice as analerotic traits was made by Freud and confirmed by others. Ambition, envy and impatience were regarded as urethral character traits and some of the same traits were traced by Abraham to the oral stage of libido development. Freud studied the character stage at the menopause, when a sweet loving mother changes to a petty, miserly, peevish old lady. Abraham's study of the female castration complex was another analytic contribution to the understanding of character. These earlier formulations were cemented together in Freud's "Das Ich und das Es" which postulates three systems in the development of the personality: (1) the id, which is the great reservoir of instinctive and repressed activities; (2) the ego, which constitutes the highly modified and conscious part of the id; (3) the super ego or ego-ideal, which is the resultant of the struggle with the oedipus situation and represents a climax in the development of character processes. The activities at this level are represented by the imperative "thou shalt" and "thou shalt not." The individual mode of reacting to these commands, at the different periods of this ego and libido development, determines the character pattern found on him and also the prevailing mode of gratification. The case of the man who was constantly getting himself involved in money affairs and indulged in financial self punishment is an example where character traits of a pathologic stamp were the resultant of a thwarting during the genital and pregenital development.

In explaining the neurotic person's ability to preserve his sense of reality, which the psychotic has lost in the formation of a delusion, attention is called to the two stages of libidinal development. The first or auto-erotic and autoplastic period, where instinct tension is met by auto-erotic methods, and the second or more adult alloplastic period where tension is relieved by modifying the environment. The neurotic has remained at the forbidden auto-erotic level, and the neurotic symptom disguises the fact. Real gratification is given up and the sense of reality is retained. This compromise represents a socially acceptable modification of his character, and a rigid system of rationalization carried on through all the waking hours maintains its efficiency. Ferenczi regards these as private psychoses tolerated by the ego.

The neurotic character makes demands on the environment, and then sees to it that they are not fulfilled and that the man actually receives punishment from striving to achieve them.

Knowledge of factors leading to the formation of the neurotic character has assisted in making more exact formulation concerning normal character processes, which are defined as a "set of organized behavior tendencies founded on and tending to preserve a stable equilibrium between id tendencies and submission to reality."

ALLEN, Philadelphia.

A Theory of Decussation. Ian D. Suttie, J. Neurol. & Psychopath. **6**:267 (Feb.) 1926.

In view of the apparent complication of the central nervous system by decussations, inquiry is permissible as to what countervailing advantage is thus conferred. There is no functional evolutionary interpretation of the crossed central system. It cannot be for the purpose of integrating the two halves of the body. Crossed sticks are just as separate as parallel ones. Evidence suggests that the central nervous system was originally uncrossed and that the crossing is connected with the location of stimuli. In the optic apparatus the number of uncrossed fibers is at a minimum when the eyes are laterally placed, at maximum when visual fields cross the midline. This might be for the purpose of maintaining the functional heterolaterality of the sensorimotor centers. But if lateral eyes and a complete decussation at the chiasma, as in fishes, represent the primitive arrangement, this would not explain why there is a crossing; it merely explains why there is a partial crossing where fields of vision overlap. The author suggests that the optical arrangement present at the evolutionary inception of the crossed nervous system might have been that of parallel axes or even of mesial eyes. The eye is unique as a sense organ, as the lens reverses the image of the environment and so transposes the stimuli before they affect the nervous system. The retinas are thus affected by a reversed environment. Connections must then be evolved with the motor centers on the other side of the body, and centers must be built up for the correlation of optical data with those from the different sense organs, also mainly on the other side.

But why are these correlation centers and the higher motor centers built up beside the optical centers — that is to say, crossed? The process of cephalization itself must have been largely determined by the influence of optic stimuli, and thus the higher sensorimotor centers are built up, not only at the head end of the cord, but also on the opposite side from their peripheral connections. It is therefore suggested as a purely theoretical possibility that the decussation of the central nervous system is a response, to a considerable extent adaptive, to the reversal of images produced by a lens-containing eye whose field of vision crosses the middle line.

If this theory is valid, it would lead one to imagine the early evolution of vertebrates as follows. First there was a wormlike creature, swimming or crawling at the bottom of relatively shallow water; then dorsal cephalic eyes developed, either median or paired. In the latter cases, the optic axes are still roughly vertical and, therefore, their field of vision overlaps the midline. At first they might be without muscles. At most, a primitive musculature would be able to direct the eye or eyes to one side or the other. A diagram is given showing how the simplest possible reflex arc to direct the eye toward a source of stimulation, by means of a muscle inserted in the superficial half of the sphere of the eye, requires an association fiber crossing in the midline of the nervous system. (Four typographic errors in the text disagree with the diagram.)

An attempt is made to explain the unique decussation of the trochlearis by assuming that two eyes originally developed mesially, in series, on the neural crest, and that subsequently they shifted to become paired organs. A second mesial ophthalmic rudiment exists in lampreys. Thus the muscle, which at first deviated the mesial eye toward its own side, is now itself on the opposite side of the body; but its nerve has its central attachment as formerly. The trochlearis nerve of the right eye thus represents the innervation of the left muscle of that eye when it was median. It might be conjectured that the rest

of the neuromuscular mechanism is evolved in the side to which the eye now wholly belongs and largely from the original oculomotor apparatus of that side and eye.

The function of the original oculomotor (third) nerve would be to deviate both eyes toward its own side. One branch of each nerve would be carried across the middle line and function would not be greatly disturbed.

The author states that, obviously, his theory will require a great deal of evidential corroboration before being accepted. He leaves this to others. Two possible difficulties, however, are dealt with: (1) A chiasma formed in the manner described would be dorsal to the neuraxis. It is suggested that the present chiasma really is dorsal in an evolutionary sense and that the infundibulum, which develops at the site of the neuropore, represents the anterior pole of the neuraxis while the cerebrum is a dorsal upgrowth. (2) Why should vertebrate (lateral and frontal) eyes have supplanted invertebrate (pineal and paraphyseal)? A solution involves the suggestion that when the bottom-swimming protovertebrate becomes free-swimming it requires lateral and ventral more than dorsal vision. Thus the chiasma shifts cephalad, and the development of the dorsal cephalic nuclei compels a ventral flexion of the anterior end of the central nervous system.

FAVILL, Chicago.

HYPERKINESIA OF ORGANIC AND PSYCHOGENIC ORIGIN. ROBERT BING, Schweiz. Arch. f. Neurol. u. Psychiat. **18**:163, 1926.

Study of the extrapyramidal syndrome has been of great interest to neurologists, especially in regard to the types of disturbance that seem to form a link between organic affections and psychologic states. Epidemic encephalitis has produced many syndromes of hyperkinesia which only a few years ago were classified as being of purely psychogenic origin. As examples may be quoted the tics seen in postencephalitic Parkinson's syndrome and respiratory disturbances, such as paroxysmal sighing, sobbing and yawning, as well as crises of involuntary howling. The spasmodic elevation of the eyes, torticollis and myoclonic movements of the limbs also come into this category. Bing remarks on the striking similarity of so many of the descriptions taken from a vast number of case reports, and comments on the stereotyped similarity which seems to exist between a large number of cases. Some of the patients observed in the remote portions of Europe resemble identically those seen in other portions equally remote.

Not only is it possible for these hyperkinesias to be seen in cases of epidemic encephalitis, but they may appear in other pathologic conditions having the subcortical ganglia as the center of attack. Areas of softening, degenerations and syphilis have produced tics, myoclonus and spasms of the muscles similar in character to those seen in epidemic encephalitis. The question therefore comes up as to whether the spasms and tics previously called idiopathic and considered psychic in origin may not have some pathologic basis, especially in view of the meager knowledge concerning the pathologic changes that might be present in the basal ganglia. So many of these patients have been classified as psychopathic and hysterical, especially since they had a definite neuropathic heredity and since many of these motor phenomenon have followed psychic injuries and emotional upsets. On the other hand, in the more severe cases of facial spasm, spasmodic torticollis and similar conditions, the most patient and painstaking psychologic investigations may show little that might be invoked as causal. In many of these patients, somatic and mental stigmas of hysteria are absent, and the cases are extremely refractory to all types of

psychotherapy. While other manifestations of hysteria, even of long standing, may be rapidly cured by psychotherapy, it is only in exceptional instances that any success is obtained in the treatment of patients with these hyperkinesias,

Attempts have been made to separate clinically spasmodic movements of psychogenic origin from dyskinesias of organic disease. Frequently the organic type of movement may manifest itself in an isolated muscle that it would be impossible to innervate voluntarily. On the contrary, the tic should be a movement which it is possible to imitate voluntarily. It is, however, to be noted that from the standpoint of psychotherapy this differentiation avails little, for the treatment of patients with these tics by psychotherapy is not satisfactory, and a cure is rarely seen. Suicides are common among these patients, which is not in agreement with experience with hysterical persons, who even when their trouble appears to be extremely painful seldom seek suicide. The author quotes a case with which he had experience ten years ago, in which the patient had suffered from a facial tic; when treatment failed, he killed himself. At the autopsy too much importance was attached to some adhesions and thickening of the meninges in the region of the exit of the facial nerve, and no attempt was made to examine the basal ganglia and midbrain microscopically. Even today, after all the intensive work done on patients with encephalitis and other extrapyramidal syndromes, little has been done to establish the pathology of the more common cases of idiopathic tic or spasm. Autopsies in these cases are rare, and even in those cases in which they have been made, practically nothing has been done. These is no doubt that there is some similar pathologic process underlying the so-called idiopathic tics and choreo-athetosis and post-encephalitic involuntary movements.

PARKER, Rochester, Minn.

MICROSCOPIC OBSERVATIONS IN IDIOTYPIC LATERAL SCLEROSIS (SPASTIC HEREDODEGENERATION). CARL SCHAFFER, Arch. Psychiat. u. Neurol. **77**:675 (Aug.), 1926.

This paper contains a discussion of the diseases of the heredodegenerative system, with a report of the pathologic conditions in a case of spastic lateral sclerosis. The clinical picture was described in a previous communication together with a clinicopathologic study of the patient's brother, who presented a somewhat similar picture. In both cases the onset occurred at about the age of 3 years and was accompanied by spasticity in the legs, which gradually spread to the arms, without any disturbances in sensibility or control of the bladder. The younger brother died at the age of 28 of pulmonary tuberculosis, whereas the man whose case is now reported died at the age of 38. In contradistinction to the first patient, the elder brother showed more marked spasticity, changes in the eyegrounds in the form of temporal pallor and bilateral central choroiditis, slight atrophy of the musculature of the hand, but more marked atrophy of the triceps and tibialis anterior (as in amyotrophic lateral sclerosis) and tremor of the head and outstretched hands. There were no psychic disturbances.

The postmortem examination showed:

(1) *The Pyramidal System.*—In the anterior central convolution, which was the only one affected in the whole telencephalon, was a degenerative process showing varying degrees of affection in the different layers. The ganglion layer showed a simple dropping out of cells. The pyramidal and multiform layer showed, besides this, the Alzheimer type of fibril change (similar to the type found in presenile conditions). In the third and sixth layers there were signs of swelling of all apical, and of some of the basal and lateral, dendrons. The pyramidal tracts

apparently were normal throughout their course above the cord, and only the myelo-architectonics of the precentral convolution showed slight changes, being less deeply stained in Weigert preparations.

(2) The next important change was found in the cells of the anterior horn in the cord. Here, only in the region of the fifth lumbar segment, definite changes in the form of swelling of the cells of the anterior horn, with distinct signs of neuronophagia and some slight fat infiltration, were found. Stains of the myelin sheath showed degenerative processes in the pyramidal tracts from the thoracic segments downward. A similar process was also found in the tracts of Goll on both sides, which was probably directly dependent on the changes found in the spinal ganglia. These showed: (*a*) signs of faulty development in the form of twin cells and bi-axonal ganglion cells and (*b*) a progressive degenerative process beginning with fenestrations and ending in total destruction of the cells. Whereas the developmental dysplasia was present throughout all segments, signs of definite progressive degeneration were found in the lumbosacral regions only, which would explain the fact that only the Goll tracts showed signs of degeneration, the Burdach tracts apparently being intact. The tremors suggested possible changes in the extrapyramidal system, but outside of the macroscopic apparent atrophy of the caudate nucleus, no histologic changes could be found. Similar to the signs of developmental dysplasia in the spinal ganglia, there were double nucleated pyramidal cells in the precentral convolution, and superficially one could see a definite anthropoid-like fissure formed by a peculiar configuration of the parieto-occipital and intraparietal fissures.

There were no signs of infiltration or other disturbances of the vessel walls throughout the brain and cord. The pia and arachnoid were apparently normal, so that the mesodermal elements seemed to have taken no part in the disease process.

The author concludes that in this case of heredodegenerative disease, one deals with: (1) a process that shows special predilection for ectodermal tissue; (2) one that shows predilection for certain systems, viz.: (*a*) the upper and lower motor neuron system and (*b*) the spinal ganglion-ascending tract system leaving all other segments apparently intact. The constitutional nature of the process is suggested by its occurrence in two members of the same family and by the signs of developmental dysplasia in both the brain and the cord.

MALAMUD, Foxborough, Mass.

IDIOPATHIC NARCOLEPSY: A DISEASE SUI GENERIS; WITH REMARKS ON THE MECHANISM OF SLEEP. W. J. ADIE, Brain **49**:257 (Sept.) 1926.

Idiopathic narcolepsy, first described by Gelineau, is regarded by the author as a separate and distinct disease. It is characterized by two kinds of attacks — sleep without cause, and attacks brought on by emotion called cataplexy. It occurs in both sexes and is a disease of adolescence and early adult life. The frequency of the sleep attacks varies from two or three a day to one every few minutes, but is fairly constant in each person. Sleep may occur under any kind of circumstances, favorable or unfavorable, and the attack is always inevitable, resembling in every particular normal sleep. The sleep may be light or profound, but any kind of external stimulus will arouse the sleeper.

The second kind of attack is produced by some form of emotion, laughing or anger, during which the patient loses control of the muscles and falls to the ground, but does not lose consciousness. These symptoms are attributed to loss of tone in the muscles; the head drops forward, the arms drop to the sides, the knees give way, and the person falls, unable to move or talk. The patient recovers quickly as a rule, but a sleep attack may immediately follow. These

attacks are called by the author cataplexy. He regards cataplexy and sleep attacks as due to the same underlying mechanism and, therefore, they are both expressions of the same process, differing only in degree.

Fifteen cases from the literature together with five personal ones are described in this paper, and the disease is differentiated from epilepsy, hysteria and other disorders of which narcolepsy is a secondary manifestation. In the latter case the author insists the term symptomatic should be used, leaving the term idiopathic for the cases of true narcolepsy which occur in otherwise healthy persons.

Pawlow's conception of sleep is discussed in detail, and his conclusion that sleep and internal inhibition are one and the same process are in accord with that of the author following his application of this theory to the sleep and cataplexy of narcolepsy. Monotonous stimuli favor attacks of sleep as the receptive point in the cortex becomes fatigued, producing localized sleep, and inhibition spreads unchecked over the cerebral cortex and to subcortical centers to produce general sleep. Extraneous stimuli when present will prevent sleep. In the words of the author, "true narcolepsy is a functional disorder of the nervous system, probably an undue fatigability of nerve cells, in individuals with a peculiar kind of nervous activity that allows excessive responses to emotional stimuli and favors the spread of inhibitions." Two cases resembling true narcolepsy following epidemic encephalitis were presented, but in these cases there was always improvement, whereas idiopathic narcolepsy persists throughout life.

The author postulates a subcortical center in the interbrain which sends out inhibitory impulses by nervous paths, thereby inhibiting cortical activity. It influences sleep but by what mechanism is not known. He believes that narcolepsy is primarily a disorder of the pituitary interbrain system, an endocrine-nervous mechanism, composed of the pituitary body, the nucleus hypophyseus and adjacent vegetative centers in the floor of the interbrain. Diabetes insipidus, dystrophia adiposogenitalis and other endocrine disorders along with narcolepsy may arise from this system; idiopathic narcolepsy is produced from the interbrain centers, whereas symptomatic narcolepsy of pituitary origin arises from lesions of that gland. A long discussion of sleep centers is presented with special references to the theory of Economo who believes the centers are situated in the interbrain and midbrain, as it is in these areas that the pathologic process is most pronounced in cases of epidemic encephalitis in which sleep and ocular palsies were the predominating symptoms.

The author maintains that sleep may arise in the cortex or in subcortical centers, but the latter should not be referred to as the sleep centers. The subcortical center can influence sleep primarily, but it is only a part of the mechanism of sleep which is controlled not by single centers but by the balanced action of many nervous and endocrine nervous mechanisms working in unison.

STACK, Milwaukee.

ABLATION EXPERIMENTS ON THE LABYRINTH OF FROGS. JOHN TAIT, Arch. Otolaryng. **4**:281 (Oct.) 1926.

The writer quotes experiments on frogs illustrating the fact that the semicircular canals influence the dynamic equilibrium, and the utricle, static equilibrium. No influence on equilibrium was noticed in frogs when the saccule was removed or the saccular nerves cut. The limbs and body of the animals were observed, with no attempt to study nystagmus. He concludes that the utricle is wholly an organ of static equilibrium. Static equilibrium depends on: (1) pressure

impulses due to contact with the ground, (2) impulses from muscles and joints, (3) impulses from the eyes and (4) impulses from the utricular maculae. When the utricular macula of one side is destroyed, and the animal is placed on a horizontal surface, it leans to the side on which operation has been performed. Rotation has no effect, whereas tilting of the horizontal plane causes the animal to assume a new posture. Frogs were decanalized without disturbing the utricle and found to have lost their dynamic equilibrium, as evidenced by their response to quick movements. They had perfect control when the movements took place slowly, showing that the utricle was unaffected. They found that they were able to diagnose individual lesions of any of the four vertical canals in the frog by observing the movements of the limbs. The author illustrates the function of the utricle and of the canals with various ingenious models. A frog in which the right horizontal canal was destroyed, failed to step to the left when rotated quickly to the right, showing that quick rotation to the right in a frog stimulated the right horizontal canal. The same was proved for pigeons by Ewald and has been confirmed in other animals. Land vertebrates walk on four struts or legs, and in the usual form of progression the body is supported alternately on pairs, consisting of right fore and left hind, and of left fore and right hind respectively. In each case the girder bridging the upper end of the limbs passes diagonally across the trunk, and the body may be said to swing in each case about a diagonal axis. Now, with respect to the vertical canals, the right anterior and the left posterior can be considered as constituting a parallel pair, while the left anterior and right posterior constitute another parallel pair. The right anterior vertical and the left posterior vertical canals are in the same plane. One vertical canal was destroyed on the frog tested. The experiment was repeated on other frogs until all the canals had been studied, leading to the conclusion that: "Each vertical canal is especially associated with the limb of that quarter of the body toward which it points. A vertical canal is stimulated when the head is suddenly tipped downward toward the quarter to which the canal points. The main effect of stimulation of a vertical canal is a sudden extension of its own particular limb, which is thrown diagonally away from the body, that is, further toward its own corner. In this way the limb is prepared to exert a thrust (1) upward, against the weight of the falling body, (2) horizontally, in the direction toward the center of the body. We may also note that whereas a vertical canal is specially associated with one particular limb, stimulation of a horizontal canal leads to movements of at least two, and usually of all four limbs." The canals may be divided into two groups, the horizontals or nongravity, concerned with waltzing movements, and a gravity set, concerned in leaping and landing on the ground. A tilt-table was constructed and various patients examined, and the results noted. These cases will be reported on when a sufficient number have been studied.

HUNTER, Philadelphia.

THE AFFECTIVE SYMPTOMATOLOGY OF DISSEMINATED SCLEROSIS. S. S. COTTRELL and KINNIER WILSON, J. Neurol. & Psychopath. **7:1** (July) 1926.

Although the term "mental symptoms" has been used frequently by previous writers to denote changes in the emotional or intellectual sphere, an attempt at explanation or at differentiation of the actual symptoms has not been made heretofore. From the results of the present paper, it is evident that a change in the emotional reaction is almost universal, while intellectual changes are negligible.

The following cardinal symptoms belonging to the emotional or affective sphere are the result of an analysis of 100 consecutive cases and are independent

of degree, duration or neurologic type of the disease. In all of the patients there has been a change in mood; 63 per cent exhibited a frank emotional euphoria, 10 per cent an emotional dysphoria, and 25 per cent an increased variability of the mood. The second cardinal symptom, that of exaggeration of the emotional expression, was seen in 95 per cent of the patients with disseminated sclerosis, 87 per cent of them being aware of it. A sense of physical well-being, eutonia sclerotica, was present in 84 per cent of the cases, while in only 6 per cent was there a physical dystonia. Even in cases with pronounced advancement of the neurologic process, cases in which the patients are bedridden, this sense of physical well-being was most pronounced. This condition is in no way dependent on the euphoria; it is more prevalent than that symptom. To the aforementioned symptoms must be added another, that of optimism (spes sclerotica) which is independent of any of the others. This was present in 84 per cent of the patients, 15 per cent only being pessimistic in their outlook.

These affective symptoms are characteristic of the disease; they are primary or direct results of the disease process, and completely independent of duration, degree, or clinical type. In many cases they precede the appearance of any somatic neurologic symptom, and are regarded by the authors as constituting a diagnostic triad of greater value than any neurologic symptom-complex.

In discussing the pathogenesis of these affective symptoms the authors believe that the emotional overaction, occurring so early and constantly in the course of the disease, is somewhat associated with dysfunction of the paleothalamus and that this is attributable to early invasion of that structure or its association tracts by the toxin of the disease acting via the ventricular ependyma. Owing to the frequent invasion of the thalamus by the pathologic process and its rare appearance in the cortex, it is reasonable to assume that the exaggerated sense of bodily feeling is the result of misinterpretation of incoming impressions at, and from, the level of the optic thalamus. A probable localization for emotional mood exists in a special center, its cells being specific for that function only, situated in one of the basal areas affected as a result of ventricular invasion. Applying their results to other fields the authors believe that certain psychoses and psychoneuroses characterized by changes in the affective field have a toxic or toxistructural and not a psychopathologic basis. STACK, Milwaukee.

THE SUGAR METABOLISM IN CHRONIC EPIDEMIC ENCEPHALITIS AND PARALYSIS AGITANS. R. A. TKATSCHEW and W. W. AXENOW, Ztschr f. d. ges. Neurol. u. Psychiat. **104**:391 (Sept.) 1926.

Claude Bernard first demonstrated the fact that injury of the floor of the fourth ventricle causes hyperglycemia and glycosuria. More recently Brugsch, Dresel and Levy pointed out that the area punctured by Bernard corresponded to the dorsal nucleus of the vagus. Injury of the middle and caudal portion of this area caused hypoglycemia, while injury of the anterior or cephalic portion caused hyperglycemia. Brugsch, Dresel and Levy found two vegetative centers in the medulla: one, the sympathetic, which innervated the suprarenals, and the other, the parasympathetic, which innervated the pancreas. By studies in secondary degeneration they were able to demonstrate a center in the nucleus periventricularis which regulates the work of the medullary centers and maintains the sugar content in the blood. They demonstrated, moreover, a higher center localized in the pallidum, and were able to show changes therein in diabetes mellitus. Spatz and Lubarsch, however, consider the observations of these authors on the pallidum of diabetic persons as being wholly within normal limits.

Although Brugsch, Dresel and Levy defend their views with few facts, they at least offer a new point of view. Experimental and clinical work recently has shown that important vegetative centers lie in the midbrain; centers for albumin, fat, and water metabolism and for temperature lie there. Moreover, the histologic structure of the cell groups in the midbrain — nuclei supraopticus, paramedianus, paraventricularis, substantia grisea — is similar to that of the sympathetic cells.

Tkatschew and Axenow studied the sugar metabolism in cases of chronic encephalitis and in paralysis agitans on the basis of the work of Brugsch, Dresel and Levy. Their patients were assumed to have disease of the basal ganglia and midbrain. Fasting sugar contents of the blood and urine were determined in their cases, from 50 to 100 Gm. of dextrose were given, and the sugar content of blood and urine was studied for the next five hours. The microchemical method of Bang was used in the determinations. Thirteen cases of postencephalitic parkinsonism, six cases of morbus parkinsoni, and one case of double athetosis were studied. According to the authors' interpretation, all thirteen cases of postencephalitic parkinsonism showed disturbance in the sugar metabolism. In plotting the sugar curves, a return to normal was not found after five hours in one case, a return to normal was found during the fifth hour in two cases and during the third hour in five cases. In one case the blood sugar rose to 255. In four of six cases of Parkinson's disease a disturbance was found. Return to the normal level occurred in the fourth hour in the case of double athetosis. In their cases of postencephalitic parkinsonism they demonstrate a marked disturbance in eight instances, slight in three cases, and normal in two.

ALPERS, Philadelphia.

BLADDER DISTURBANCES IN LESIONS OF THE NERVOUS SYSTEM. I. LEON MEYERS, J. Nerv. & Ment. Dis. **64**:321 (Oct.) 1926.

Bladder disturbances are of two types: retention, and incontinence of urine. It is not known whether retention is caused by failure of expulsive powers of the bladder or by overaction of the sphincter. Retention of urine and straining at urination, as well as frequency of urination, constitute the initial type of disturbances of the bladder occurring in all lesions of the spinal cord when the seat of the lesion is above the conus. This disturbance of the bladder is due to the destruction of the fiber tracts which form the vesical pathways on their way to and from the cerebrum. These tracts can offer considerable resistance to a slowly developing destructive process, and disturbance of the bladder is generally a late symptom. In lesions of the conus or of the cauda equina, retention of urine is the initial type of disturbance when the lesion causes paralysis of the pelvic autonomic nervous system, the posterior horns and roots, but does not involve the motor nuclei of the anterior roots of the internal pudic. Lesions of the conus or cauda equina, associated from the outset with involvement of the motor nuclei of the internal pudic or its anterior roots, may have passive incontinence as the early manifestation of bladder dysfunction. The nuclei of origin in the conus, as well as of the roots forming the cauda, appear to be less resistant than the fiber tracts of the bladder and the spinal cord.

Retention, whether the result of lesions of the spinal cord or conus, may or may not be associated with incontinence or overflow. It is generally succeeded sooner or later, by passive incontinence, which is the terminal state of dysfunction in hopeless cases. In some cases, retention may be followed by the so-called automatic bladder or active incontinence. Retention of urine is one

of the earliest manifestations of tabes dorsalis. Retention is the result of failure of expulsive powers of the bladder, owing to the paralysis of the pelvic autonomic nervous system and the detrusor muscle, the escape of urine by gravity being prevented by the mechanical action of the musculature of the perineum which keeps the urethra compressed. In complete transverse lesions of the cord, the sympathetic nervous system of the bladder is paralyzed simultaneously with the pelvic autonomic nervous system, and does not function independently of the cord by virtue of its ganglia in the mesenteric plexus. Such paralysis is undoubtedly associated with the relaxation of the internal sphincter, and probably hastens the development of incontinence. It does not prevent the urine from accumulating in the bladder so long as the musculature of the perineum is in good condition.

HART, Greenwich, Conn.

CONTRIBUTION TO THE ANATOMY AND FUNCTIONS OF THE HYPOPHYSIS. II. THE NERVOUS REGULATION SYSTEM OF THE POSTERIOR LOBE OF THE HYPOPHYSIS. THE NUCLEUS SUPRA-OPTICUS AND ITS FIBER SYSTEMS. R. GREVING, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:466 (Sept.) 1926.

In 1922, Greving described the fiber systems of certain cell groups, among which was the nucleus supra-opticus. Besides the tractus supra-opticus superior, which formed a part of the lower thalamus, he described the tractus supra-opticus inferior. According to his description then, this tract passed around the medial side of the optic tract to the tuber cinereum, where it was lost between the cells of the substantia grisea centralis and the cells of the tuber cinereum.

In 1925, he demonstrated a fiber bundle which ran from the vicinity of the nucleus supra-opticus through the tuber cinereum into the infundibulum and posterior lobe of the hypophysis. This tract he designated as the tractus supra-opticus-hypophyseus; it is identical with the tract described, in 1922, as the tractus supra-opticus inferior. J. L .Pines, in 1925, confirmed these observations. Greving attempts in this present contribution to determine more about this tract, and to find out if other fiber systems are connected with the nucleus supra-opticus. He concludes that: (1) The nucleus supra-opticus surrounds the optic tract at the foot of the midbrain, and that it consists of cell groups, a pars dorsolateralis, dorsomedialis and ventromedialis. These cell groups, however, are connected with one another and form a single cell area which extends down as far as the tuber cinereum. In this way one can understand why Lewy obtained pathologic cell changes in the tuber cinereum after a lesion of the posterior pituitary lobe. (2) From the large cell territory of the nucleus supra-opticus there arises a large fiber bundle, the tractus supra-opticus-hypophyseus, which passes through the tuber cinereum to the infundibulum to enter the posterior lobe of the pituitary. "The assertion of Lewy, therefore, of a connection between the posterior lobe of the hypophysis and the nucleus supra-opticus is demonstrated in serial sections in man anatomically." (3) At the cell area of the nucleus supra-opticus certain fibers are acquired which come from the gray matter of the third ventricle. These arise from the nucleus paraventricularis from the vicinity of the chiasm almost to the commissura intermedia. This tract is designated as the tractus paraventricularis-cinereus. It ends in the vicinity of the nucleus supra-opticus. (4) The nervous regulation of the posterior lobe of the hypophysis arises, therefore, from the nucleus paraventricularis, tractus paraventricularis-cinereus, nucleus supraopticus, and the tractus supra-opticus-hypophyseus.

ALPERS, Philadelphia.

MULTIPLE NEURITIS WITH FACIAL DIPLEGIA. ALBERT YUDELSON, J. Nerv. & Ment. Dis. **65**:30 (Jan.) 1927.

Although the incidence of facial diplegia in multiple neuritis is generally considered as unusual, Patrick collected thirty-one cases, and recorded the condition as due to a virus affecting the peripheral nerves, ganglia and spinal cord — a view no doubt generally supported. Holmes studied twelve cases in 1917. Patients complain of malaise, pains in the legs and lumbosacral spine, motor weakness of the lower extremities, paresis of the arms, bilateral weakness of the face and disturbance of speech and deglutition. The temperature ranged from 102 to 103 F. The deep reflexes were absent early, but sensation was seldom impaired. Two of the twelve patients died. Bradford recorded thirty cases of acute infectious polyneuritis in 1917. A slight illness from five to six weeks prior to the palsy was characteristic. Pyrexia was slight. Paresthesias were followed by motor weakness. Facial diplegia was practically constant in Bradford's series. In fourteen of seventeen cases it was bilateral; recovery was slow, taking about six months. Pathologic observations by Bashford show minute, widely diffuse hemorrhages in the dorsal enlargement, proliferation of ependymal cells of the central canal and irregular reduction of nerve cells in the anterior horns. The condition was evidently a septicemia entering the nervous system by way of the nerve trunks, thus passing to the ganglia and the spinal cord. Bashford transmitted the disease from man to monkey and from monkey to monkey, with symptoms and pathologic processes identical with those in man. The organism is an anaerobe of diploid, reniform appearance.

The author regards the frequency of the involvement of the seventh nerve in this case as being due to the aptitude of the infection to invade motor nerves, the seventh having the largest purely motor distribution of any of the cranial nerves. The author presents a case of typical polyneuritis in which facial diplegia occurred in the course of the ascent of the infection. Almost complete recovery was attained about seven months after the onset.

HART, Greenwich, Conn.

RESECTION OF THE SUPERIOR CERVICAL SYMPATHETIC GANGLION IN A CASE OF FACIAL PARALYSIS. BOTREAU-ROUSSEL, Arch. franco-belges de chir. **29**: 732, 1926.

Resection of the superior cervical sympathetic ganglion has been performed in facial paralysis a number of times, chiefly for the relief of epiphora and lagophthalmos. The author reports a case in which the patient was wounded by a pistol ball traversing the head from the left side of the nose to the right mastoid region, fracturing the petrous portion of the temporal bone. Cerebrospinal fluid ran from the ear. The patient experienced coma, total facial paralysis on the right and a slow pulse; his general condition was poor. The patient recovered sufficiently under expectant treatment to be operated on forty-eight hours later. At this time the bony spicules were removed, and it was found that the ball had glanced upward after fracturing the petrous portion. The dura was sutured after exploration failed to reveal the projectile. A small fistula required another operation, but recovery was complete in 1923. Facial paralysis was marked, with epiphora and lagophthalmos. In the beginning of 1924, he had mild jacksonian attacks, with a disagreeable vertiginous sensation and sometimes loss of consciousness. In February, 1925, the superior cervical ganglion was removed. Immediately after this, the upper lid fell, and it seemed that the

mouth was less drawn to the left. While the patient was still unconscious, the right (paralyzed) cheek was tickled and the corner of the mouth was drawn to the right. At the present time the face is much more symmetrical. The lagophthalmos and epiphora have disappeared. The eye can be closed practically completely, and the patient can carry out certain voluntary movements with the mouth, being able to eat on that side. In talking or laughing, the mouth still deviates somewhat to the left. He is unable to wrinkle the forehead. No circulatory disorders have been noted. The right pupil is somewhat contracted. There have been no further jacksonian attacks. The explanation of this return of voluntary power, following cervical sympathectomy, is not given by the author.

FREEMAN, Washington, D. C.

THE THEORY AND PRACTICE OF PERMEABILITY TESTS BY THE "BROMMETHOD." K. WALTER, Arch. f. Psychiat. u. Nervenk. **79**:363 (Jan.) 1927.

The author, the originator of the method, discusses the various aspects of permeability tests, especially with reference to the work done by Buchler (reviewed in ARCH. NEUROL. & PSYCHIAT. **17**:673 [May] 1927). There is no doubt as to the existence of a semipermeable membrane between the blood and spinal fluid. The permeability, although not purely physicochemical, lends itself to physicochemical tests. Different substances, especially salts of the different metals, behave like the bromides, that is, after ingestion they are found in greater quantity in the blood than in the cerebrospinal fluid. The bromides are preferred because they can be recognized with greater ease, and the proportion in the fluid and blood is relatively constant.

When investigating changes in permeability there are several sources of error to be considered: the type of colorimeter used, the concentration of the bromides in the blood and cerebrospinal fluid, and the amount of gold chloride used. The author works with high concentrations (about 1 in 3,000), because then the gold chloride, which is slightly brownish itself, is used up in the formation of gold bromide. He does not find the degree of decrease in permeability in dementia praecox recorded by Buchler. Buchler, in 252 cases found ten permeability quotients between 5 and 8, whereas the author found none in that range in 800 cases. Buchler's statement that the permeability is normally low in young persons and high in old age is, according to the author, not supported by investigations by himself and others. Walter consequently believes that increased or decreased permeability is indicative of a pathologic rather than of a physiologic change in the barrier. He concludes that the permeability tests can be used with other symptoms for diagnostic purposes. There is definite increase in permeability in general paralysis and in arteriosclerosis, and definite decrease in postencephalitic conditions. The symptoms in functional psychoses are not sufficiently definite for diagnostic purposes.

MALAMUD, Foxborough, Mass.

JUVENILE PARESIS. ITS SALIENT CHARACTERISTICS, WITH SPECIAL REFERENCE TO INFANTILISM. HENRY A. BUNKER, JR., Am. J. Syph. **10**:553, 1926.

Since the recognition of juvenile general paralysis by Clouston, in 1887, between 275 and 300 cases have been reported in the literature. Approximately 1.5 per cent of the children with congenital syphilis develop general paralysis, as compared to 5 per cent of the adults with syphilis. The distribution of the juvenile form between the sexes is nearly even, whereas the ratio of females to males in cases among adults has been variously estimated as 1 : 2, or 1 : 4. Other differences are a

longer duration in the juvenile form, with more frequent occurrence of optic atrophy, ataxia, attacks of vomiting, convulsive seizures, hemiplegia and palsy of the cranial nerve. Retarded growth is more characteristic of juvenile general paralysis than of any other form of congenital syphilis, and this retardation is nearly always manifested by underdevelopment of the whole person rather than of particular parts. The author reports a case showing typical features. The father had acquired syphilis nine years before the patient's birth. The patient was a full term child, weighing 7 pounds (3.2 Kg.). There was no history of convulsions, but growth was retarded, and at the age of 14 he began to show mental changes. At 16, he gave up his work, complained of difficulty of vision, showed a tendency to fabrication, ataxia, defect in speech and marked irritability, which later gave place to drowsiness. On admission to the hospital he had a spinal fluid cell count of 19, increased globulin, a general paralytic gold curve, and a positive Wassermann reaction in both the blood and the spinal fluid. He showed marked spontaneity, circumstantiality and frequent lapses in speech. The intelligence quotient was 91. Both pupils were irregular and did not react to light or in accommodation. There was slight left ptosis and a left external strabismus. The tongue deviated to the left. The Romberg test showed slight swaying. The patient was underweight and below the normal height for his age, but the ratio of weight to height was nearly normal. This patient did not respond to malaria treatment except by a slight modification of the Wassermann reaction.

JENKINS, Philadelphia.

A CONTRIBUTION TO THE STUDY OF DEPOSITS CONTAINING CALCIUM AND IRON IN THE BRAIN. ELIZABETH COWPER EAVES, Brain **49**:307 (Sept.) 1926.

The author states that hematoxylin is not a suitable stain for testing for calcium in the central nervous system as other salts give similar reactions, and an abundant amount of calcium may fail to produce a reaction if it occurs as an acid salt. No microchemical method gives undeniable proof, for which the chemical method following ashing of a part of the brain must be relied on. Two cases of angioma of the brain, characterized clinically by epileptic seizures, were studied pathologically; in one the pathologic process was most pronounced over the occipital lobe, in the other it occurred intensely in the corpora striata and diffusely in other areas of the brain. The deposits consisted of a gritty material which on unstained sections contained many concentric masses, or concretions, similar to the so-called "psammoma" bodies. None contained pigment; many contained iron; in others it was entirely absent to the Prussian-blue test.

Chemical examination of both cases, following ashing of a small piece of the brain, showed that the deposits contain both calcium and iron, the former as phosphate and carbonate, the latter partly as phosphate and partly as some other insoluble compound not hemosiderin. Calcium salts are soluble in hot acetic acid while the iron salts are insoluble. In both these cases, iron and calcium were found in great abundance above the amount normal in the brain. In a case of epidemic encephalitis, in which these same globules were abundant in the midbrain, many of the smaller ones gave the iron test while other similar ones did not. In this case iron was found to be present in the walls of the blood vessels of the dentate nucleus, but only a small amount was present in the corpus striatum. In five of six cases of general paralysis, iron-containing pigment was found in the walls of the blood vessels in the cerebral cortex, but it differed from that found in the above cases, resembling hemosiderin in staining reactions and solubility.

The question of the formation of calcium and iron in nerve tissue, along with the significance of the presence of one influencing the formation of the other, is discussed, but no positive answer can be deduced from results so far obtained.

STACK, Milwaukee.

ANGIOSPASMS OF THE CENTRAL NERVOUS SYSTEM—AN ATTEMPT AT CLASSIFICATION. F. BREMER and H. COPPEZ, J. de neurol. et de psychiat. **26**:563 (Nov.) 1926.

The authors recall the fact that cerebral arteries lack a vasomotor innervation which renders the explanation of cerebral angiospasm obscure. However, it must be added that, although the cerebral arteries do not react to pharmacodynamic substances that influence the vegetative nervous system, they may react to mechanical or chemical agents which presumably are the cause of the pathologic angiospasm. The classification which the authors propose is: (1) Angiospasms due to a local lesion of the vascular walls (atheroma or specific arteritis). This is by far the most frequent cause and the best known. (2) Angiospasms associated with the Raynaud syndrome, the determination of which is still unknown, its pathogenesis being complex. 3. Angiospasms due to exogenous intoxications (quinine, nicotine and others). (4) Angiospasms due to endogenous intoxications (Bright's disease, essential hypertonia, hemicrania and perhaps essential epilepsy). (5) Nervous angiospasms. This category is provisional and includes cases in which no other appreciable causes may be found and in which the patient proves to be hyperemotive. A reflex spasm of one of the basilar vessels, which still has vasomotor innervation, may explain the global phenomena following hypermotivity.

The symptoms due to the angiospasm are the result of consequent ischemia in the central nervous system and are first irritative and then paralytic in nature. When the pathogenesis is toxic or nervous, the ischemia does not last long. Symptomatic treatment is still the best we have and is based on vasodilator and antispasmodic effects of benzyle benzoate as well as on the action of phenobarbital given in fractional doses.

FERRARO, New York.

FATIGUE: A CLINICAL STUDY. R. D. GILLESPIE, J. Neurol. & Psychopath. **7**:97 (Oct.) 1926.

There is no satisfactory definition for fatigue. It has been defined as a "diminished capacity for work." This has the disadvantage of being *a priori*, since only by working to the point of exhaustion can this fatigue be fully demonstrated. The following clinical formula is convenient: A person is fatigued when he is capable of less general activity than is usual with him, or when he is capable of less activity than would be expected from his general mental and physical equipment, and when the diminution in activity is accompanied by a sensation of fatigue, exhaustion or weakness. Fatigue may be investigated by observing the method of performance of the organism in whole or in part, and by observation of isolated phenomena such as tremor or blood reaction. The theories advanced for the etiology of fatigue syndromes can be conveniently classed in five divisions. These are constitution, autointoxication, excessive effort, emotion and the psychoanalytic theories. Fourteen case histories are given in detail. These and certain other cases seem to fall naturally into the following five groups: (1) cases in which fatigue is the direct expression of a conflict; (2) cases in which fatigue is a prodromal symptom of serious mental illness; (3) cases in which fatigue accompanies other symptoms which together make a picture of a depression or of a schizophrenic psychosis;

(4) cases in which fatigue may be a symptomatic equivalent to a recurrent depression, (5) and cases in which fatigue may be a sequel of a variety of mental disorders.

Several methods of treatment of fatigue are suggested. The principal rôle must be assigned to psychotherapy. Graded exercise can increase tolerance. Hydrotherapy is useful in improving vasomotor tone. Exercise can increase vital capacity. The administration of phosphates, formerly empirical, now has a sound basis.

FAVILL, Chicago.

DIAGNOSIS AND TREATMENT OF SYPHILIS OF THE CENTRAL NERVOUS SYSTEM. EDWARD LIVINGSTON HUNT, Am. J. Syph. **10**:537, 1926.

The most constant symptoms of syphilis of the central nervous system are irregularity, inequality and immobility of the pupils. Premature aging, personal carelessness and failure of judgment and memory are significant. It should be borne in mind that the premonitory symptoms may often be mistaken for neurasthenia. The author holds that involvement of the nervous system occurs earlier than is generally believed, and probably within the first weeks following infection. The reason for the apparent selectivity of certain strains of *Spirochaeta pallida* for the nervous system remains undetermined. The examination of the spinal fluid is the most instructive aid in diagnosis and the results are rarely negative in a neurosyphilitic patient. The cell count is of primary importance, and the colloidal gold curve second, but unless the curve is definitely that of general paralysis or tabes, it is of questionable value. Emphasis is placed on the frequent presence of a negative blood Wassermann reaction in tabes, as well as on the variability of the reaction from day to day in the same patient. Treatment is briefly considered under the headings specific and nonspecific. Bismuth, a marked spirocheticide, is of low toxicity and gives good results in Wassermann-fast cases, while tryparsamide is a less efficient spirocheticide and depends more on an influence on metabolism for its effect. In the treatment of general paralysis with malaria, marked remissions occur in one third of the cases, a fatal termination in 10 per cent, and in less than 50 per cent no change is seen. The most favorable cases for this treatment are those of middle-aged men in the early stages of the disease. Failure to regain the weight lost during the chills is an unfavorable prognostic sign.

JENKINS, Philadelphia.

THE ANALYSIS OF SPINAL FLUID TESTS. JAMES B. AYER, J. A. M. A. **87**:377 (Aug. 7) 1926.

Ayer believes that observations on the spinal fluid often do not yield as much value as they might, that the chief significance of the fluid tests is missed and that, occasionally, misinterpretation leads to wholly erroneous conclusions. While Ayer states that inadequacy of laboratory facilities is occasionally at fault, he believes that too many are concerned with the fluid, and that a puncture, an adequate number of tests, and an analysis of the fluid by the physician who knows the clinical history will yield a maximum of information.

Ayer enumerates the tests that yield information of the greatest value as follows: (1) initial pressure and pressure studies; (2) naked eye study for color, turbidity, clotting and blood, three successive tubes being used; (3) the total number and differential count of cells; (4) a general protein precipitant and if practicable a quantitative determination; (5) the globulin test with ammonium sulphate; (6) the Wassermann test; (7) the gold chloride or benzoin test; (8) direct smears and cultures, and rarely inoculation; (9) quantitative estimations of sugar, chlorides and nonprotein nitrogen.

In support of his point that the simplest tests carried out and analyzed by the physician who performs the puncture and also knows the patient are of the greatest value, Ayer describes five cases that illustrate also the necessity of intellectual effort in interpreting the tests. He also emphasizes the importance of negative tests when positive tests had been expected.

CHAMBERS, Syracuse, N. Y.

THE LOCALIZATION OF THE FUNCTION OF CALCULATION. S. E. HENSCHEN, Arch. f. Psychiat. u. Nervenk. **79**:375 (Jan.) 1927.

The author discusses the article by Berger in the preceding volume of the same journal which criticizes some of the statements of Henschen. The author agrees with Berger in that calculation functions may show disturbances when other intellectual functions, such as logical thinking, are not appreciably affected, and vice versa. Berger's statement that Henschen in his monograph has singled out the angular gyrus as the calculation center is emphatically denied. Henschen states that for correct calculation function one needs normal interaction of different areas in the brain, but that some of these areas are more important than others. This depends on the fact that optic and acoustic memories and images play an important rôle. From clinical and pathologic investigations one finds that the ability to calculate correctly is closely connected with the left hemisphere, and in this especially with the occipital, parietal and temporal lobes.

In the three cases reported by Berger, the tumors were of such size and their remote effects of such complexity that, according to the author, the influence on the angular gyrus cannot be excluded with certainty. The author does not agree with Berger in the latter's statement that calculation disturbances in cases of aphasia cannot be regarded as primary.

MALAMUD, Foxborough, Mass.

THE TENDON REFLEXES OF THE ABDOMEN. TRIOUMPHOFF, Rev. neurol. **1**:307, 1927.

To elicit this reflex, the region of the pubic symphysis is percussed from 1.5 to 2 cm. from the median line. There follows a contraction of the corresponding muscles. This phenomenon differs from the mediopubic reflex described by Guillain in that the reaction is more apt to be localized to the side percussed. The reflexes seem to run parallel with the cutaneous reflexes and may be absent or weak in a person whose abdominal wall is thick or flaccid. It is not found in animals, even in the monkey. In the child the rectus abdominis tendon reflex appears at the same time as the cutaneous abdominal reflex. Occasionally in tabes the tendon reflex may be lost and the cutaneous reflex may persist. Sometimes in a mild hemiplegia this reflex is more active on the paralytic side, and in multiple sclerosis the tendon reflex is usually preserved while the cutaneous reflex is lost. Guillain says this is true in 50 per cent of the cases. In the late stages, both reflexes may be abolished.

From a biologic point of view, the abdominal tendon and the cutaneous reflexes are of the same order and have the same significance. They represent the result of the heightened tonus of the abdominal wall brought about by the assumption of the vertical position of the body. Being recent acquisitions phylogenetically, these reflexes disappear and diminish in cases of abolition of cortical influence.

FREEMAN, Washington, D. C.

OBSERVATIONS ON THE RÔLE OF THE CEREBRAL CORTEX IN THE CONTROL OF THE POSTURAL REFLEX. W. T. KING, Am. J. Physiol. **80**:311 (April) 1927.

According to King, the motor cortex in the cat has its seat in the anterior and posterior sigmoid gyri. This would appear to be so since electrical stimulation of this area is followed by appropriate motor responses and its ablation by transient paralysis. Such ablation, however, is not attended by hypertonus or essential postural abnormality. When, however, the gyrus proreus or "frontal" lobe (homologous to the intermediate precentral area of Campbell in man), which is "silent" to electrical excitation, is extirpated, striking and persistent contralateral extensor hypertonus is noted. That this reaction is dependent on specific anatomic deprivation and is not merely an irritation effect is attested by its permanence, likewise by its nonoccurrence after simple motor cortex ablation and in irritated control animals. In explanation the author regards this phenomenon as a "release" consequent to the removal of inhibition normally exercised by the frontal lobe. Whether, however, the mechanism through which this inhibition is effected is direct (pyramidal) or indirect (via basal ganglia, cerebellum or midbrain) cannot be determined at this time and remains a problem for further study. King also reaches the general conclusion, from this work, that in the cat the gyri prorei may be postulated as cortical centers of control with reference to postural relationships.

RAPHAEL, Ann Arbor, Mich.

THE MACROGLIA AND MICROGLIA IN A CASE OF GENERAL PARALYSIS. R. RODRIGUEZ-SONOZA, Trav. d. lab. d. rech. biol. d. l'Univ. d. Madrid **24**:289 (Dec.) 1926.

The neuronal changes are of two types: atrophic sclerosis and acute swelling. The former are more marked in the superficial layers of the cortex, the latter in the deeper layers. The macroglial changes show a similar topographic arrangement, being more severe in the more superficial layers; they consist of: hyperplasia, with the formation of numerous proliferative foci, and hypertrophy; increase in the density of the marginal glia. The microglia likewise shows marked reactions, again more severe in the superficial and less severe in the deeper layers; the changes are hyperplastic, hypertrophic, degenerative and destructive; there is a relative poverty in typical rod cells and absence of fat granule cells. The changes in the cortex of the cerebellum show an approximately equal degree of change, similarly distributed. The basket cells show sclerosis, while the Purkinje cells show either sclerosis or acute swelling. The alterations in the macroglia and in the microglia are similar to those in the cerebral cortex, though rod cells are found somewhat more frequently.

SINGER, Chicago.

MALIGNANT HYPERNEPHROMA WITH ARTERIOSCLEROSIS IN CHILDREN. P. R. DIETERLE, J. Nerv. & Ment. Dis. **65**:42 (Jan.) 1927.

The author presents a case of a girl, aged 4 years and 3 months, with autopsy after a history of convulsions alternating with stupor, and preceded a month previously by unilateral convulsions. The blood pressure was from 145 to 160 systolic and from 90 to 100 diastolic. Roentgenograms and operation confirmed the presence of a large irremovable tumor mass, which was diagnosed malignant nephroma on the basis of the pathologic process. Autopsy showed edema of the lungs, slight lymphoid hyperplasia, small genitalia, a small thyroid and a brain larger than normal with areas of cortical softening. The basilar

artery showed thrombus formation and thickening of the wall of the artery. The intima of the cerebral vessels showed thickening of patchy character, with heavy deposits of lipoids, and the arteries showed increase in thickness of all layers. The striking feature of the case was the coincidence of the tumor of chromaffin tissue with arteriosclerosis, arousing the theoretic discussion of the relation of the suprarenal cortex to vascular changes and development of the brain, and the analogy to Alzheimer's disease, in which arteriosclerotic processes are related to hypothyroid states.

HART, Greenwich, Conn.

A STUDY OF LUES AMONG MENTAL DEFECTIVES. FRANK R. HALL, Am. J. Syph. **10**:563, 1926.

Of 3,750 cases in the Rome State School from 1915 to 1926, forty-three or 1.15 per cent showed a positive Wassermann reaction. In eight of the forty-three a history suggestive of syphilis was found. Lesions of the eye were found in ten cases, keratitis being the most frequent. Glandular enlargement was not more frequent than in cases in which the Wassermann reactions were negative.

JENKINS, Philadelphia.

POSTENCEPHALITIC OBESITY. WALSH, J. A. M. A. **87**:305 (July 31) 1926.

Walsh directs attention to postencephalitic obesity as a clinical entity. He reports four cases and states that, in addition to these, reports from foreign literature show such close chronologic relation to the history of encephalitis that he believes a definite etiologic relationship is indicated. In the four cases reported, the rapid gain in weight and the preceding encephalitis were the outstanding clinical features. The adiposity is usually of general distribution but may be of a pituitary type. The etiologic relationship between the obesity and the encephalitis is borne out not only clinically but also by experimentally produced lesions of the midbrain. Postmortem evidence of organic pituitary injury in epidemic encephalitis is negligible and gives further weight to the neurogenic basis for postencephalitic obesity. Clinically, the differential diagnosis between postencephalitic obesity and hypopituitarism is often difficult, and specific investigation alone can bring out the encephalitic basis for the obesity, which might otherwise be attributed to simple endocrinopathy.

CHAMBERS, Syracuse, N. Y.

MODIFICATION OF VENOUS PRESSURE IN ORGANIC HEMIPLEGIAS AND FOLLOWING CEREBRAL TRAUMAS. MAURICE VILLARET and DEMÈTRE JONESCO, Presse méd. **34**:1266 (Oct. 9) 1926.

The method used to take these venous pressures, described more fully previously by one of the authors, requires venipuncture. Following that, pressure is read directly in centimeters of water; at the elbow, the normal range is between 11 and 14 cm. Arterial pressures were taken concurrently in the cases reported, and were not found to give such decisive indications as did the venous pressures. The latter were deemed particularly valuable in medicolegal cases in which symptoms suggestive of cerebral injury were otherwise not localizing and hence not conclusive; observations of venous pressure added the localization in certain cases. On the paralyzed side, venous hypertension was uniformly found in the flaccid cases of organic hemiplegia, and unilateral hypotension in the spastic cases. In the one case of old cerebral injury, without localizing signs noted, the side opposite the injury was neither spastic nor flaccid, but showed marked venous hypertension; the venous pressure of the other side remained normal.

HUDDLESON, New York.

FACIAL PARESIS AS A MANIFESTATION OF TUMOURS OF THE UPPER HALF OF THE CERVICAL SPINAL CORD. WALTER M. KRAUS and NATHANIAL E. SILVERMAN, J. Neurol. & Psychopath. **7**:132 (Oct.) 1926.

As a result of study of twelve cases, the authors conclude that paresis of the infrabuccal portions of the facial musculature may occur in association with tumors of the upper four cervical segments, and, per contra, no such paresis appears in cases of tumors of the lower four cervical segments. In two cases of a series of ten high cervical tumors which came to autopsy, one intramedullary and one extramedullary, the seventh nucleus was normal, and there was no evidence of any abnormality in its region or above the foramen magnum. In seven cases, five of which were extramedullary, from the Montefiore series, and in two cases, both extramedullary, recorded by Elsberg, this facial paresis was present. In three cases, one intramedullary and two extramedullary, from the Montefiore series, the paresis was absent. The phenomenon is probably due to disorder of a reflex pathway originating in this portion of the spinal cord and extending to the facial nucleus.

FAVILL, Chicago.

CORTICAL HETEROTOPIA IN THE PONTILE MENINGES. WALTER FREEMAN, Arch. Path. & Lab. Med. **2**:352 (Sept.) 1926.

The author reports a case of a colored woman, who died of pneumonia in the course of an acute toxic psychosis. At necropsy, a small, flattened, oval circumscribed mass, lying wholly within the meninges, was found over the upper end of the pons on the right side. Grossly, the mass resembled normal cortex and was not invading nor connected with any nerves. Microscopically, the tissue resembled normal cortex, its cyto-architecture being closely allied to that of the hippocampal area. The fibers ran either tangentially or perpendicularly, but straight. Only rarely did a myelin sheath occur. The glia was normal. There were two distinct sets of meninges, one for the pons, the other for the heterotopia. There were no other malformations in the brain. The author was unable to find a report of a similar case in the literature.

PEARSON, Philadelphia.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, March 25, 1927

M. A. BURNS, M.D., *in the Chair*

THE EVOLUTION OF OUR KNOWLEDGE OF THE BRAIN DURING THE LAST SIXTY YEARS: ILLUSTRATED WITH A SERIES OF PERSONAL OBSERVATIONS. DR. CHARLES K. MILLS.

This paper is intended to be largely autobiographic. One of its chief designs is to show that any large subdivision of the brain cannot be understood without taking its anatomic and physiologic relations to other parts of the same great organ into consideration. I entered on my neurologic experiences with some handicaps and some advantages. I did not have a master to guide and counsel me when I started on my neurologic career, nor did I have, like some of our distinguished colleagues of this Society, the advantages of foreign study. On the other hand, I had command of abundant clinical and pathologic material, at an early date, as the result of my connection with the neurologic services of the University of Pennsylvania and the wards for patients with nervous diseases of the Philadelphia Hospital. I shall try to show how my personal development, as illustrated by the cases described and authorities referred to, was largely that of the evolution of neurology during the last sixty years.

At the last meeting of this Society a somewhat acrid debate was precipitated regarding the prefrontal lobes as anatomically and physiologically a higher, or the highest, psychic subdivision of the brain. Some of the speakers seemed to think that to recognize a higher or highest psychic area in the brain was equivalent to locating the mind in this situation. This is a serious, but not uncommon, mistake. One part of the brain may be the organ of the mind or of mentation, if this term is preferred, but the anatomic boundaries of the mind itself are much larger and may, in fact, take in the entire nervous system, or, as some have suggested, the entire body. Evolution enters into the decision of such a question.

According to Hughlings Jackson—and I do not know of any one better for a neurologist to follow—the nervous system is divided into at least three levels, possibly more. The lowest, first and best organized is that of the spinal cord; the second or middle level evolves from the first and includes the motor and sensory areas of the cerebrum; the highest level, which represents the final evolution of the nervous system, has its abiding place in the most anterior, that is, the prefrontal, portion of the brain (*J. Ment. Sc.* **33**:25 [April] 1887).

The question of centers comes into a discussion of this sort. Most would-be critics have a false idea of what a localizationist means when he speaks of brain centers. Here again, I believe, Hughlings Jackson's definition is the best, and it is the one to which I have always adhered. A center, according to Jackson, is simply a spot or location in the brain in which a particular movement or impression is represented in greater measure than anywhere else. Every such center is associated in some way with other parts of the brain, as all parts are connected or related anatomically and functionally (Evolution and Dissolution of the Nervous System, London, John Bale and Son, 1888, p. 1).

In the process of evolution the highest psychic prefrontal region, as it evolves more and more, becomes more strongly organized, and eventually is nearly independent of the lower levels from which it has been developed. In apparent contradiction to the general law that the last organized is the least organized, this highest evolutional level, taken as a whole, is the most organized. Within this highest level however internal evolution is constantly taking place or tending to take place, and, speaking only of this level, the last organized becomes the most evanescent or the least organized.

It is within this region that such higher qualities of the mind as will, memory, reason and emotion are represented; they are not represented by limited and separated units or centers, but by the working together of the highest and the most complex nervous machinery here represented. This region is an area of what Jackson terms re-re-representation, in which the nervous levels below for both sensation and emotion are triply represented (*J. Ment. Sc.* **1** [April] 1887).

It happens that some of my first published neurologic experiences were related to the prefrontal lobe. One of the first cases of tumor of the brain which came to necropsy, which I observed and reported, involved the first and second frontal, the convolution of the callosum and the callosum itself of the right side of the brain. I published this case in detail (*Philadelphia M. Times* **9**:184 [Jan. 18] 1879). The mental symptoms, briefly told, were: slowness of comprehension, lack of attention, explosive speech, loss of memory and lack of reasoning faculties, such as comparison and judgment, and emotional manifestations, including weeping.

Since recording this case I have had somewhat numerous midfrontal and prefrontal cases under my observation, accounts of some of which have been published, like that reported by Dr. Weisenburg and myself (*J. A. M. A.* **46**:337 [Feb. 3] 1906). The symptoms, although differing in minor respects, were all examples of tract impairment or loss of the higher attributes of mentality.

In my experience, cases in which the lesions in the brain were on the right side, were particularly noticeable for emotional manifestations. Emotion and emotional expression take the same place in extent of representation in the right cerebral hemisphere that language does in the left.

This is as good a place as any to recall my contributions on the subject of emotion and emotional expression. I first became interested in these questions from observing patients in the wards for nervous diseases at the Philadelphia Hospital who exhibited a variety of emotional manifestations which were not under control of their wills. Sometimes these manifestations were apparently of a distressing sort, as weeping with or without lacrimation; sometimes they were agreeable in appearance, as involuntary laughing, smiling or grinning. In the course of time I had the opportunity to make necropsies in cases of this sort which I had studied during life. I found lesions variously distributed, as for instance in the cortex, in the lenticular, in the caudatum, in the geniculate bundles of the internal capsule, in the corticobulbar tracts, in the nucleus ruber, in the pons oblongata, in the cerebellar prepeduncle or in the thalamus. I thoroughly discussed the subject of the cerebral mechanism of emotional expression in two papers, one presented at a meeting of the College of Physicians in 1911 (*Tr. Coll. Phys.*, Phila. **34**:147, 1912), and the other at the meeting of the American Medico-Psychological Association, May 28-31, 1912 (*Tr. Am. Medico-Psycho.* **19**:297, 1912). The paper presented to the College of Physicians was afterward published in a memorial volume to Bianchi.

I will present, in condensed form, a few of the most important facts and hypotheses included in these papers. I held that emotion and emotional expression were separately represented in the cerebral mantle, the former in the prefrontal

region, where, as Bianchi (A Textbook of Psychiatry for Physicians and Students, New York, William Wood & Company, 1906) and others have maintained, the final syntheses occur which result in the higher psychic processes including emotion, while emotional expression is more especially represented in the midfrontal region, in a zone closely contiguous to that concerned with emotion itself. I had shown that an emotive zone representative of emotional expression was probably located in the midfrontal and posterior part of the prefrontal region, that zone being entirely cephalad of the motor zone as usually regarded, that is, forward of the precentral convolution. This zone also included an anterior projection of the second and upper portion of the third frontal convolution, and it was more highly developed in the right hemicerebrum than in the left.

The views maintained by me were derived partly from the results of faradic excitation of the human cortex during operations by Dr. Frazier (*Univ. Penn. M. Bull.*, Phila., **18**:134 [July and Aug.] 1905), and partly from a general study of the literature on the subject, psychologic and neurologic. Among the movements demonstrated as having their representation in the midfrontal zone for emotional expression were those for closing and opening the eyes, for opening the mouth, both by movements of the jaw and of the lips, for bending the head forward, backward or to one side, and for the movements of Darwin's (The Expressions of the Emotions in Man and Animals, London, John Murray, 1872) so-called muscles of grief. These muscles, especially in the upper part of the face, are for the movements of the corrugator supercilii, the frontalis, the pyramidalis nasi and the muscles of the eyelids (*Tr. Coll. Phys.*, Phila., **34**:147, 1912).

Hughlings Jackson, broadly generalizing, applies the evolutional theories of Herbert Spencer, Laycock, Bain and others to the explanation of increasingly complex reflexes as we pass from the lowest to the middle and from the middle to the highest levels of the brain. He lays down the proposition that the frontal half of the cerebrum is motor and the posterior half is sensory. I appreciated this fact early, and in 1888 I indicated my conviction that the sensory region was not only separate, but was situated posteriorly to the rolandic fissure (*Tr. Cong. Am. Phys. & Surg.*, 1888, vol. 1; *Brain* **12**:233 [July] 1889; ibid. **2**:258 [Oct.] 1889).

One object of this paper has been to show that the evolution of our knowledge of the brain, and especially of localized centers in the encephalon, during the last sixty years, has been illustrated by my own clinical and pathologic experiences. I have recorded cases authenticated by operations and necropsies occupying almost every particular functionally limited districts of the brain—cases of prefrontal, midfrontal, postfrontal, postcentral, postparietal superior and inferior, occipital, third frontal, insular, retro-insular, supratemporal, midtemporal, thalamic, subthalamic, superior, inferior and posterior cerebellar and spinal. An excellent opportunity of rounding out my experience in cerebral localization by the report of an actual case was afforded and put on record in a paper by me, "The Cerebral Centers for Taste and Smell and the Uncinate Group of Fits" (*J. A. M. A.* **51**:879 [Sept. 12] 1905).

This paper recorded a case in which a tumor, arising in the uncinate convolution, spread to other regions of the brain. I had notes of this case extending back to 1899 and 1900. I can only briefly give its main features.

The patient became subject to epileptic seizures differing from one another in some details but having as marked features distinct auras of smell and taste with smacking of the lips and champing of the jaws; he died at the close of a serious seizure. Necropsy showed a tumor which evidently had begun in the uncinate region and had extended backward and inward.

My personal views on inhibition differ somewhat from those usually held. It is well known to all that, in cases of incomplete transverse lesion of the spinal cord, reflexes in the parts supplied below the lesion are exaggerated, sometimes grossly so, as evidenced by such phenomena as patellar clonus, ankle clonus and the toe reflexes of Sinkler (The Toe Reflex, *Med. News* **53**:611 [Dec. 1] 1888), and other defensive reflexes. Usually these cases are explained on the theory of withdrawal of cerebral inhibition, but I doubt the correctness of this explanation.

In a paper presented to the Philadelphia County Medical Society, in 1915 (*Penn. M. J.* **18**:496 [March] 1915), I cited Bastian's (*Medico Chirurgical Trans.* **73**:151, 1890) well known case of complete transverse lesion of the cervical cord, and referred to another case of my own of equally complete transverse lesion of the thoracic cord. Certainly all cerebral inhibition must have been withdrawn in these cases; yet in both cases and in others which I have studied, there was complete atonic paralysis with lost reflexes in the lower limbs.

My view is that there is a special peripherospinal and cerebral tonectic apparatus (*Neurol. Centralbl.* **33**:1266 [Dec. 16] 1914). Sensory stimuli maintain a standard tonectic state in the normal person or a uniform condition of tonic innervation. When the corticotonectic stimuli are rhythmically delivered, they maintain the motor system in the state of normal muscle tonicity. Complete withdrawal may occur either through lesion of the sensory pathway or through more or less complete lesion of the pyramidal tract. If there is a partial block, the result is an overcharge of tonic innervation. The hypertonia is expended in a forcible and irregular way on the lower spinal neurons and gives rise to the resulting hypertonicity and increased reflexes.

It is at least doubtful whether the Nissl second type of small cells is set aside for inhibition. My own studies, of the cerebral cortex, of the striatum and of the cerebellar deep nuclei, point to these small cells as associating in function. In fact, to me it does not seem necessary to assume two types of cells, one for excitation and one for inhibition. The large motor cell types may exert inhibitory influence in particular cases, as can be shown by numerous examples. Inhibition is a function of much importance in the central nervous system, but it does not follow that it requires a distinct type of cells for its performance, as in the vegetative system.

In 1917, a paper by Dr. George Riddoch (*Brain* **40**:265 [Nov.] 1917) appeared on "The Reflex Functions of the Completely Divided Spinal Cord in Man, Compared with Those Associated with Less Severe Lesions." In 1920, a paper by Head and Riddoch also appeared (Studies in Neurology, London, Oxford University Press, vol. 2, 1920, p. 467). The work by Riddoch was conducted largely under the direction of Head. Both of these articles have great significance in connection with the discussion of inhibition.

Riddoch's paper at first seemed to oppose Bastian's views, founded on his cases of complete division of the spinal cord in the cervical region, and also my views founded on cases of cervical and of thoracic complete transection. Careful reading of the paper, especially of the cases reported by Riddoch, indicates that Riddoch's observations and conclusions are not entirely opposed to those of Dr. Bastian and myself.

The question of the origin of cerebral tone must first be borne in mind. Tone, like every other function in the human nervous system, has its origin in the outside or inside periphery. Impressions or stimuli in the periphery outside the body are received by exteroceptors, namely cutaneous and mucous membrane receptors. Impressions or stimuli from within the body—from muscles, joints, bones and

labyrinth—received by propioceptors are conveyed with the cutaneous sensory impressions to the thalamus and thence to the sensory tonectomotor cortex and lastly to the highest or psychic prefrontal regions.

The peripheral impressions or stimuli are received and transformed in the cerebral tonectic zone and these transformed impulses are conveyed by efferent tracts, pyramidal and extrapyramidal, to the centers in the oblongata and spinal cord.

The cerebellum is, in one sense, a combination of the lowest spinal level and the middle level. It is subordinate to the cerebral level of which it is a sort of appendage or appanage. Tone derived from the periphery is primarily a cerebral function. Tone, however, may be aroused directly in the spinal cord or in the spinocerebellar portion of the cerebrospinal apparatus.

Inhibition is an active process. When all the levels of the nervous system are intact, tone impulses or stimuli are conveyed to all parts of the motor nervous system and keep all movements in a state of normal balance. If the spinal cord is severed in the cervical or thoracic region, tonic innervation is no longer conveyed from above to the spinal centers below the site of the section; hence atony results, and the muscles, tendons, etc., become flaccid, and the reflexes disappear or are greatly lowered. In the first place, this is the result of the shock of an operation or injury completely severing the cord. When the shock passes off, reflex activity begins to return through the influences or stimuli received directly from the periphery. In this way we have the phenomena of the reflex activity described by Head and Riddoch. These reflexes, however, never attain the normal perfection of reflexes which are present when the spinal cord is intact.

In cases of partial lesion, cases in which the cord is incompletely divided, tonectic impulses force their way through the imperfect block and overcharge the centers below the site of the lesion.

The discussion of the reflex activities of the bladder and the phenomena of sweating after complete spinal transection are particularly instructive. Head and Riddoch quote largely in defense of their position from Sherrington (The Integrative Action of the Nervous System, London, 1906) and also from Jackson.

NOTE.—This paper, as first presented to the Society, included a somewhat elaborate discussion of the cerebellum and also of sensation and its cortical representation. The cerebellar material has since been included in a paper presented at the annual meeting of the American Neurological Association in May, 1927, and the notes on sensation have been expanded into a "Discussion of Sensory Disorder in Organic Disease of the Nervous System" for the combined meeting of the Neurological Section of the Royal Society of Medicine and the American Neurological Association held in London, July 26, 27 and 28, 1927. These remarks on sensation will appear in the transactions of this combined meeting to be published in *Brain*.

DISCUSSION

DR. J. H. LLOYD: Dr. Mills has taken the pains to go over practically the long period of his work as a neurologist, for which I believe we all feel grateful. In discussing the paper, I find the field so large that it is necessary for me to limit myself, and I have picked out merely one or two points to which to refer.

Dr. Mills wisely avoided discussion of the so-called faculties of the mind, and has substituted the term mentation. This is a good enough term for our purpose. The present day school of psychologists is opposed to the old-fashioned terms. They do not like the term, mental faculties. One of them would even abolish the term consciousness. They use new terms instead, such as mental complexes,

mental mechanisms, behavior patterns, correlations and integrations. What do they mean? Dr. Mills has avoided all that, and has substituted one comprehensive term—mentation.

Dr. Mills holds that the higher mental functions are located practically in the prefrontal region. He has entertained that idea for a long time and has described a syndrome caused by lesions of the prefrontal region, consisting of retarded cerebration and explosive speech. I saw a man with a fracture of the skull recently, and the roentgen ray showed that the fracture extended down into the frontal bone; the man had the exact condition Dr. Mills has described. On operation, a blood clot was found over the prefrontal region. I have seen many such cases, and the syndrome, if I may call it that, is unmistakable; but I must say that I hesitate to limit the higher mental functions to the prefrontal area.

To illustrate what I mean, I may speak of the evolution of the cortex of the brain. Herrick, in his recent book, shows that there is no cortex of the brain in fishes, and what is called the pallium is nothing but an epithelial membrane. It has no nerve elements in it except possibly three limited areas in the higher forms of fish where there may be a beginning of the cortex of the brain. One is over the big olfactory structures and becomes the hippocampus, and the next is over the corpus striatum. These two constitute the first development, the archipallium. Then there is another extending a little forward called the neopallium, from which is evolved the cortex of the higher forms of life. As we ascend the vertebrate scale, through the reptiles, birds and lower mammals, this cortex grows larger and larger in the neopallium, and the fair inference is that the function called mentation grows with it. Especially in those regions of the cortex which are the centers for the great cranial nerves it is likely that this function develops; in other words, the higher mental faculties are probably very active in these centers. For instance, many of our ideas depend on our use of language; it has even been debated whether we could form abstract ideas and engage in abstract thinking without the use of language. Now this center is in the temporal lobe (largely), in the auditory centers, the primary center for speech. Hence I infer that some of the higher mental functions may be active here.

The same may be said of the visual centers in the occipital cortex, for many of our higher and more complex mental processes are connected with the sense of sight; and also of the superior parietal lobule, where is located the stereognostic sense, by which we have many of our goemetric ideas.

From all this I should infer that the higher functions of mentation are not limited to one region of the cortex. Nevertheless, I think, with Dr. Mills, that they manifest themselves in a peculiarly active and dominant way in the prefrontal areas.

In discussing the cerebellum, I refer again to comparative anatomy. The cerebellum in the reptile is exceedingly small, according to Edinger, but in the birds it has undergone an enormous evolution. It is almost as big as the forebrain. A bird is endowed with an important function—the function of flight—which requires the finest adjustment, the finest balancing. It does seem, therefore, that the cerebellum in birds has something to do with flight. All we know about the human cerebellum we have derived from the study of its pathology. That is not the best way to study a thing, but we are forced to do it. I have seen a number of cases of lesions of the cerebellum. I saw a man who had a tumor of the cerebellum involving the middle cerebellar peduncle. He had forced movements of the head to one side, and when he sat up his body was forced to one side. It was not a hemiplegic condition or loss of power on one side, but it was an excess of power on one side. I think that tends to confirm the idea that the cerebellum

has to do with balancing movements. The same can be implied from the disease known as Friedreich's ataxia. The movements are not the same as those of tabes. They suggest a cerebellar lesion. I presented a case here a few years ago in which careful studies were made. The boy died of an acute disease, and we had the cord in the full bloom of the disease. The cells in Clarke's column at many levels of the cord were destroyed, and the direct cerebellar tracts were sclerosed. The inference was that there was interference with the cerebellar function.

Some of the cases which Dr. Mills refers to are those of the pons and associated pontile and cerebellar disease. The trouble with these lesions, it seems to me, is that they are often too destructive. No two cases are alike. One pontile lesion destroys one part and another lesion another; hence the necessity for careful interpretation, such as he gives. For instance, Dr. Mills has referred to the subject of paralysis of conjugate movements of the eyes. Some years ago I had a case of a young man who had pseudobulbar palsy. Autopsy showed lesions in the corpus striatum. That man had complete paralysis of the conjugate upward movements of the eyes, and paralysis of convergence. If I had been guided by that symptom, I should have been misled, for instead of a true ophthalmoplegia it was what some German writer has called a pseudo-ophthalmoplegia. The lesions in the corpus striatum cut off the tracts from the motor centers in the forebrain to the nuclei of the third nerve in the midbrain.

Dr. Mills has properly called attention to the necessity of differentiating between astereognosis, caused by a lesion in the parietal lobe, and cerebellar ataxia. It is also necessary to distinguish between labyrinthine vertigo and cerebellar ataxia. They are distinct; labyrinthine vertigo is subjective; there is no motor disturbance, such as ataxia or forced movements.

To me the great value of Dr. Mill's paper is its suggestiveness. It gives many hints and indications, based on a long and extensive experience, which should point the way to further fruitful study. It is the work of a pioneer and leader.

Dr. Francis X. Dercum: During the fifty years that I have known Dr. Mills, I have listened to many of his papers. Dr. Mills has not kept pace with the evolution of our knowledge of cerebral localization, he has led it. Indeed, he has been the world's greatest leader in this field. While some of our friends on the other side of the water might dispute this statement, we on this side have no hesitation in acclaiming it.

I have also listened to Dr. Lloyd's remarks, which have been delivered with his usual clearness and impressiveness. I fully share his views regarding the "behaviorists." There is only one psychology, and that is the physiology of the brain. Dr. Lloyd has approached the subject from the biologic point of view, and this also inspires my sympathy. In my early days, the nervous system of man seemed to me to be hopelessly complicated, and I turned to the fishes in which the fundamental problems of vertebrate structures are presented in a much simpler form. In the fish, the motor organ of the brain is represented by the striatum; indeed, by a structure corresponding to our own globus pallidus. Because of its primitive and relatively simple character, it is spoken of as the paleostriatum. Immediately back of this motor organ is a sensory structure, the primitive thalamus or paleothalamus. It is made up of nuclear aggregations, corresponding to those in our own epithalamus (habenula), of nuclear aggregations in the hypothalamus (eminences back of the optic decussation and the mammillary bodies) and in nuclear aggregations for smell, for taste, for the viscera and for the body generally.

There are also numerous receptors grouped about the head and spread over the body which receive the impacts from the outside world. The first impacts are the chemical impacts which are received by receptors for smell and taste and are transmitted to a large brain mass, the "nose brain" as Herrick terms it, and to the contiguous area for taste. In a similar manner, the light impacts are received by special receptors which transmit impressions to the "eye brain"; next are receptors which transmit impulses to the "ear brain"; other receptors transmit impulses to the "visceral brain"; others still, diffused over the body, transmit impulses to a "skin brain." To this primitive arrangement, Edinger applied the term paleo-encephalon. It corresponds in mammals to the brain stem, which is made up of the medulla, the quadrigeminal bodies and crura, the thalamus and the striatum. The brain stem is also spoken of as the segmental brain, because, like the spinal cord, it is capable of a segmental interpretation.

The impacts which are received by the various receptors of the fish are transmitted through the striatum to the motor neurons of the spinal segments. The fibers descending from the striatum constitute the first or primitive motor pathway. It is the only motor pathway in the fish and practically the only motor pathway in amphibians, reptiles and birds. Its fibers correspond to the pallido-rubrospinal tract in ourselves (von Monakow's bundle).

It is interesting to note that in the fish the impacts received by the receptors result in alternate contractions of the muscles of the two halves of the body; the two halves contract in sequence, and this results in the act of swimming. It is important, further, to emphasize the fact that the synaptic relations of the motor neurons of one side of the striatum of the fish are in synaptic relations with the motor neurons of the opposite side; that is, the motor pathways decussate. Here is the first instance of the occurrence of decussation and also of an explanation of the decussation; for if the motor pathway did not decussate, the contraction of the muscle would be on the same side as the impulses which leave the striatum, and forward motion of the animal would be impossible. I will return in a moment to the fact of the contraction of the two halves of the muscle masses in sequence. It is the first instance—if the action of the mandibles and fins is omitted—of coordinated movement.

This primitive nervous system of the fish answers every purpose so long as the organism remains a fish and so long as it is limited to an aquatic habitat. Changes, however, become necessary when the animal becomes terrestrial and is forced to make new adaptations, adaptations necessitating new and more complex movements. It is exceedingly probable that the neuron associations in the primitive brain are fixed; that is, they permit little or no variation or adaptation in their responses. Especially is this true of fishes and doubtless also of amphibians. In reptiles there is possibly a slight variation in responses and possibly a little more in birds. It is not unlikely that the striatum, increasing a little as the organism advanced in the vertebrate scale, primitively had a little power of changing the associations among its neurons. However, when terrestrial life was definitely established, an increase in the number of neurons took place. This increase was brought about by the proliferation, the heaping up, of intercalary neurons at the distal extremity of the neural tube. This new formation, barely noticeable, if present, in fishes, slightly more noticeable in amphibians, a little more evident in reptiles and still more evident in birds, is spoken of as the pallium; it constitutes the end-brain and in mammals becomes the cerebral cortex. The intercalary neurons of which it is composed present no fixed relationships with each other; therefore variability and an increased power of adaptation to the responses received from without become possible. These intercalary neurons gradually increase in number and promote to an

immeasurable degree the possibilities of the adaptations of the organism to the demands of a new and changing environment. Their number increases rapidly as one advances in the vertebrate scale until in man the enormous number of approximately 10,000 million is reached.

The cortex is a great usurper. With its establishment, the striatum undergoes a reduction in importance. In mammals the striatum is no longer, if indeed it has ever been, the seat of any variable or adaptable neuron combinations; but now it becomes definitely the seat merely of fixed neuron associations. It should be borne in mind, however, that the striatum increases greatly in size as the vertebrate scale is ascended; not only is the putamen added, but also the caudatum; together these structures are often spoken of as the neostriatum. The addition of the putamen and caudatum increases both the number and the complexity of the possible neuron associations. This increase corresponds to and keeps pace with the constantly increasing size and complexity of the cortex.

With the continued growth and development of the telencephalon or cortex, a new motor pathway develops, the cortical or pyramidal motor pathway, which enters into direct relations with the motor neurons of the segmental brain and of the spinal segments. There is, therefore, a double innervation in the muscles; first, the primitive innervation from the striatum through the pallidorubrospinal tract, and secondly, another innervation from the cortex through the pyramidal tract. This is entirely in accord with the interpretation which Dr. Mills has advanced of a double innervation from the upper and lower levels of the brain.

I shall now consider the most interesting feature of the entire problem. Many years ago, I became interested in the lateral line system of fishes, which consists of tubular formations containing at intervals structures which are morphologically and practically identical with the maculae acousticae of the semicircular canals. These lateral lines are distributed over the head and along the sides of the body. The interesting point is that the nerves that arise from the maculae which they contain terminate in the same area in the medulla as the nerves from the receptors in the ampullae of the semicircular canals; i. e., the auditory nerve and the lateral nerves terminate in an acousticolateral area on the posterior aspect of the medulla. Now, just as the telencephalon, the cortex, is produced by a proliferation of cells at the distal end of the primitive neural tube, so is the cerebellum produced by a proliferation of cells from this acousticolateral area. The heaping up of these cells forms the primitive cerebellum; the cells first develop structures corresponding to the nucleus dentatus and the other nuclear masses of the cerebellum. This acousticolateral area receives the proprioceptive impacts from the muscles and joints, the exteroceptive impacts from the semicircular canals, and, in fishes, also from the lateral lines. As a result of the constant inpouring of these impulses there is a constant outpouring of impulses, a continuous stream, which finds its exit from the cerebellum through the red nucleus and thence by the rubrospinal pathway to the cord and to the muscles. As a result of this outpouring of impulses, every muscle of the body is constantly kept tense like a violin string. Sometimes the pitch, the intensity of the muscle tone is raised, sometimes it is lowered, but it never ceases except in disease. In addition, therefore, to the muscle tone, due, first, to the pallidorubral innervation and secondly, to the pyramidal innervation, there is a third tonic innervation derived from the cerebellum.

Further, it is a significant fact that the cerebellum develops in proportion to the development of the telencephalon; the more the telencephalon grows, the more the cerebellum grows. Birds offer an apparent exception to this

statement, because of the relatively large size of the cerebellum, but in birds, while the pallium is small and the cerebellum large, the striatum is exceedingly large and still discharges some of the functions which are discharged in the mammalia by the usurping telencephalon.

What is the physiologic interpretation of the interaction of these three intoning processes? Impacts from the outside world received through the various exteroceptors reach the thalamus; thence they are transmitted to the cortex. Transmission through the cortex then takes place and in due course a motor exit is reached, and a "voluntary" movement is initiated. At this instant, however, there is immediate cooperation by the striatum—associated movements as in the arms and legs in walking—and also most important modifications in the cerebellar intoning, in accordance with which the tone of the muscles concerned in a given movement rises or falls.

The action of the cerebellum can be readily illustrated by simple flexion and extension of the arm. The muscles concerned both in flexion and extension are, through the influence of the cerebellum, constantly maintained in a state of tone, i. e., in a state of moderate contraction. Like the strings of a violin, both flexors and extensors are attuned to a certain pitch. When impulses are sent down from the motor area of the cortex to the extensor muscles, these muscles contract and the arm is extended, but the flexor muscles do not suddenly relax; they yield gradually like a band of tense rubber, and the extension of the limb is accomplished smoothly and evenly, not jerkily and suddenly. This applies to the extension, the unfolding of the various segments of the arm in their entirety; the extension of the upper part of the arm on the shoulder, the extension of the forearm on the arm, of the wrist on the forearm, of the hand on the wrist and of the fingers on the hand. These movements occur in an associated sequence. The separate and incoordinate extension of the segments of a limb means asynergia and is a symptom of cerebellar disease. The simultaneous and combined action of flexors and extensors, it may be added, have been termed by Tilney cocontraction.

The rôle of the semicircular canals, as Dr. Mills has stated, is most important in relation to posture and equilibration. The lateral line organs of fishes, of which I made a personal study which was published in the proceedings of the Academy of Natural Sciences in 1879, are sensory structures for the perception of succussions and coarse waves in the water, and also for the environmental relations of the fish as to depth and position in the water. In the same year I made a study of the morphology of the semicircular canals, which was published in the *American Naturalist*. In addition to the lateral lines, the fish also presents well developed semicircular canals which add to the perceptions derived from the lateral lines, those of spatial relations. It is exceedingly probable, that the semicircular canals have their origin in the lateral line organs of primitive fishes. Time will not permit a full discussion of this subject, but it is certain that the semicircular canals with their peculiar relations to each other in the three dimensions of space did not originate spontaneously. In some of the primitive and lowly organized fishes, the myxines for instance, there is only one semicircular canal; in the lamprey eel, again there are only two. It would seem that in the myxines and lamprey eels and other lowly organized fishes now no longer extant, some of the lateral line elements became deepseated and were taken in at the base of the skull of the organism by a process of involution of the surface tissues. This is the more probable as the petrosal bone in which the semicircular canals are embedded is not a part of the mammalian endoskeleton but is really an ectoskeletal structure. However this may be, the facts of the relation of the wonderful receptors in the maculae

acousticae in the ampullae of the semicircular canals are of the utmost interest The impacts received from the hair cells are transmitted to the bipolar neurons in the vestibular ganglion. Thence they are transmitted by the vestibular nerve to the group of closely related vestibular nuclei in the medulla. These vestibular nuclei in turn have most important and significant connections; namely, with the dentate and other nuclei of the cerebellum, with the various segments of the spinal cord, with the nuclei of the abducens, pathetic, trigeminal and oculomotor nuclei, with the thalamus and with the cortex. That impulses from the maculae of the semicircular canals should greatly influence posturage and equilibration, can readily be understood, while the relations to abnormalities of movements of the eyeballs, such as nystagmus, are of course well known clinically and have of late years been abundantly studied by the Bárány method.

In regard to the question which has been raised as to the function of the frontal lobes, all impulses, exteroceptive, interoceptive and proprioceptive enter the thalamus. From the thalamus they enter the cortex. Here the various sensations and feelings received from the thalamus are synthesized, and analyzed in the manner which I have described in my book on the "Physiology of Mind," and the details of which I have not the time to go into now. Suffice it to say that discrimination is the result of the purely physical processes of synthesis and analysis. Necessarily, in the posterior or sensory portions of the cortex, i. e., the portions posterior to the fissure of Rolando, thinking is concrete; one deals here with actual sensory impressions and feelings. All conceptions are concrete.

In the parts anterior to the fissure of Rolando, synthesis, analysis and discrimination are carried on to a far higher degree. Here synthesis and analysis deal with symbols, and the concrete thinking of the posterior association areas now gives way to abstract thinking. This I believe to be the function of the frontal lobes.

I wish to speak in regard to one other point only, and that is in regard to the increase of the tendon reactions in incomplete lesions of the cord and the entire loss of these reactions in complete lesions of the cord. In incomplete lesions of the cord some fibers of the pallidorubrospinal tract or of the pyramidal tract must have escaped. If the fibers of the pyramidal tract or of the pallidorubral tract have escaped, an increase of tendon reactions necessarily follows. If the cord is severed completely, all intoning influences are cut off and all tendon reactions are necessarily lost.

Dr. Mills' paper has been a great stimulant. I need hardly add that—as my remarks have shown—I am substantially in agreement with the interpretations he has given.

Dr. W. G. Spiller: Dr. Mills' paper embraces many subjects relating to the anatomy, physiology and pathology of the entire central nervous system, and he has given us gleanings from his experience covering a period of sixty years. It is difficult to know where to begin in the discussion of such a variety of topics.

He early accepted the division of the motor and sensory cortex into two separate parts, and he has seen this view gradually gain ground until now it is practically accepted by every investigator of note. Foerster believes that sensation as well as motion has a somatotopic representation in the cortex, the one corresponding in level to the other. He believes there is some representation of sensation in the precentral convolution, but this opinion seems to be based entirely on investigations carried on in the ape's brain. Dusser de Barenne has determined the extent of the sensory zone by placing strychnine on the cortex

of the ape, and has found that this zone extends into the precentral area and even into the frontal lobe. The recognition of the irritation is by means of hyperalgesia in the periphery of the body, and it is bilateral from unilateral strychnine irritation, but more intense in the contralateral limb. This method has not been used on the human brain. Foerster has used electric irritation extensively on the human cortex under local anesthesia and has never produced paresthesia from any part other than the postcentral and upper parietal areas. Strychnine is a more delicate test for sensation.

There has been doubt as to the amount of pain representation in the cerebral cortex. Foerster believes pain is so represented, but the impairment of pain disappears more quickly than does other sensory disturbance, and pain sensation usually is the least affected.

There is considerable evidence that higher psychic function is represented in the frontal lobes, but the whole brain is necessary for mental development. Two cases of tumor of the parietal or occipitoparietal lobe under my observation may be used in elucidation of this subject. These tumors, by their weight or circulatory disturbances, probably produced symptoms from the first left temporal convolution. It was possible for each patient to read words, letters and figures correctly; they made no mistakes. They found that they did not grasp fully the meaning of what they read or of what they heard. One man especially was carefully studied. He found it necessary to speak slowly and to consider the words he intended to use. This was the first evidence of a slight word deafness. He had difficulty in recalling the words he wanted, he made no mistakes, but his acoustic word center had lost some of its rapidity of action. Later he probably would have been unable to recall the word he wanted, and would have used a wrong word, and still later would have failed to understand the spoken word. A similar condition existed regarding reading. These were defects of intellect, and there was probably a mental deficit. One cannot believe that the frontal lobes can be separated from the rest of the brain and perform their function. They are connected with the entire brain by numerous tracts, the cingulum, the superior longitudinal fasciculus, the uncinate fasciculus and the fronto-occipital tract of Forel and Onufrowicz. Such extensive connection shows the importance of all parts of the brain in the proper performance of the function of the frontal lobes.

Dr. Mills has abundant proof of the effect of extensive degeneration of one cerebral hemisphere on the opposite cerebellar hemisphere. I have reported such a case, as have others.

The localization in the cerebellar cortex has been done especially by Bolk, Rijnbark and Rothmann, but there is comparatively little evidence of localization in the human cerebellar cortex. The best recent evidence of this is to be found in the work of Ingvar. With regard to the effects of complete transverse lesion of the spinal cord, I would like to direct the attention of Dr. Mills to the investigations of Head and Riddoch and others.

Dr. C. W. Burr: I could quarrel with Dr. Mills about several things. I could quarrel with him on the so-called higher psychic centers, and I would enjoy it very much. I think the cause of our quarrel would be that we are using different words to describe the same thing. That is the cause of a great number of quarrels.

Now I am in a dilemma. I cannot conceive of thought except as a chemical process, and I cannot conceive of a chemical process unless there is a place in which it occurs. On the other hand, I find it absolutely impossible to conceive what chemical process is back of wisdom, judgment and clear reasoning.

Dr. Mills: I think that those who have discussed my paper have on the whole sustained my position, even with regard to what Jackson calls the highest level of the nervous system. In this prefrontal region, nervous phenomena are triply represented—there is a region of representation in the spinal cord, of re-representation in the sensory motor middle level and of re-re-representation in the highest level. Jackson definitely speaks of the highest psychic level as concerned with memory, will, reasoning and emotion. The difficulty is that we do not all have the brain of a Jackson, a Laycock, a Bain or a Ferrier. I am certain that I do not have it; yet I understand this theory of highest triple representation.

About the question of mentation, I would like to refer to what I have written about the concrete memory field. Conceptual mentation is a sort of mentation that in one sense requires the use of higher elements of the mind. Dr. Spiller and Dr. Lloyd refer to mental loss from lesion of the temporal or parietal lobe. Number blindness, letter blindness or word blindness may occur from a focal lesion in one of these lobes. This is a form of impairment of mentation, but it is not the highest mentation or even as high as it may appear to some.

Dr. Spiller, in calling attention to the views of Bastian and myself, with regard to the persistence of atonia after complete transverse lesion of the spinal cord in the cervical or thoracic region, refers to the investigations of Head and Riddoch, with which I am not unfamiliar. These observers have shown that in a case of complete severance of the cord, after recovery from the effects of shock caused by a sudden lesion as that produced by a gunshot wound, a period of reflex activity sometimes ensues. In this period such phenomena as reflex evacuation of the bladder or of the rectum and profuse sweating in various parts of the body may occur after a strong stimulation of the sole of the foot. Other phenomena, such as flexion of the thighs on the abdomen and of the legs on the thighs, may also occur. The sweating which occurs in these cases, especially that which is seen in the parts of the body supplied by the spinal cord above the side of the lesion, is brought about through the activity of the sympathetic nervous system, the chain of the sympathetic ganglia taking their course outside of the central nervous system. The other reflex phenomena might be regarded as of a primitive sort, that is, as due to the direct influx of powerful peripheral stimuli. The permanent state of the reflexes after complete transection of the cord is one of lowering or loss.

In his discussion, Dr. Dercum, like Dr. Lloyd, gives close attention to the bearing of comparative anatomy on the question of the functions of the cerebellum. It is well known that Dr. Dercum, in the true scientific spirit from the beginning of his professional career, has based his neurologic views on his evolutional studies and on comparative anatomy and biology. It is a pleasure to me to know that these studies first made many years ago indorse the conclusions regarding the cerebellum and the entire brain at which I have arrived during my sixty years of neurologic work.

Trophic Edema of Cerebral Origin. Dr. Alfred Gordon.

Two kinds of trophic edema have been described in the literature, one by Quinke and the other by Meige. The first is acute and is characterized by rapid appearance and disappearance of swellings. The other is chronic and does not appear and reappear with the same rapidity. Both forms may have a familial character, and both may be encountered in alcoholism, saturnism, dysthyroidism, infectious diseases, epilepsy and in organic diseases of central or peripheral source. The only difference between the two is found in the fact that in the acute form the edematous fluid is rapidly absorbed, while in the chronic form the fluid persists but the sur-

rounding connective tissue is hypertrophied. The pathogenesis of circumscribed edema requires discussion. As to the fluid accumulated in the edematous areas, it is due to an obstacle in the interchange between the capillary and lymphatic systems. Clinical, physiologic and experimental facts point to the probability of an active participation of the sympathetic nervous system. On the other hand, the distribution of the edema, its segmental limitation, indicates a metameric character due to an involvement of the medulla or spinal cord as well as of the sympathetic system. In corroboration of this contention, one may cite numerous cases of diseases of the spinal cord. Anatomically, the metameric centers are associated with the sympathetic system. This fact explains the occurrence of localized edema in bulbospinal diseases. As to edema in association with cerebral lesions, only that which develops on the hemiplegic side and occupies the entire limb is found in the literature. Such a disorder is associated with the disturbance of vascular and tissue equilibrium brought on by the lesion in the brain. A large number of authors admit the existence of trophic centers in the caudate nucleus, the internal capsule, the lenticular nucleus and the lemniscus. An unusual example of trophic edema limited to one hand, "main succulente," which occurred several weeks after a cerebral insult, is presented in the following case. The cerebral insult caused only astereognosis.

A. B., aged 67, suddenly saw an object falling out of his left hand. This occurred in October, 1926. Since then he has been unable to appreciate the form, shape or consistence of an object placed in the left hand. In December, the condition was as follows: There was no paralysis, the patellar reflexes were normal, and the grip of the left hand was somewhat weaker than that of the right. Tests for sensations revealed preservation of the superficial sensibilities but considerable involvement of all deep sensibilities in the entire left hand. Astereognosis was striking. Further examination revealed evidences of generalized arteriosclerosis. Abnormalities were not found in the eyes, urine or blood. In January, 1927, the patient noticed swelling of the left hand on its dorsal and palmar aspects. The skin was not discolored or pitted. The swelling increased so that the fingers assumed a flexed position. This condition of the hand with its trophic disturbances and sensory disorders without paralysis remained unaltered until ten days prior to presentation, when the swelling of the dorsum of the hand began to decrease; at present the thickening of the skin and of the underlying tissue is pronounced only on the palmar aspect of the hand and less on the dorsum of the hand. The astereognosis remains the same, but since then there has been a gradual loss of power in the entire upper part of the left limb, thus indicating an extension of the pathologic process. The chief interest of the case lies in the trophic disorder of the hand. It reminds one of the "main succulente" observed by Marinesco in syringomyelia. It is a trophic edema of the type described by Meige. It is not the edema observed in hemiplegia, because of the absence of pitting, the color and the consistence of the tissues. This edema is not of neuritic or of bulbospinal origin, as symptoms of the latter are totally absent. It is to be presumed that the superior parietal lobe or the supramarginal gyrus, which controls the stereognostic sense, is the center for the localized edema under discussion. The case also suggests that the trophic centers are intimately associated with, and perhaps dependent on, the sensory centers, by analogy with what occurs in syringomyelia. The late development in this case, as far as the motor power is concerned, indicates that the lesion, which is probably a softening, is extending in a forward direction and affecting the corresponding motor center of the upper limb. Only the deep sensibilities were involved; touch, pain and temperature were preserved, showing that the lesion was cortical.

NEW YORK NEUROLOGICAL SOCIETY

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GEORGE H. KIRBY, M.D., *President, in the Chair*

A CASE SHOWING AN UNUSUAL TYPE OF EXTRAPYRAMIDAL DEVELOPMENT. DR. S. PHILIP GOODHART.

The patient, an only child, aged 7, was of healthy paternal and maternal stock, born at full term, weighing 7 pounds (3.2 Kg.), after a normal spontaneous delivery and without cyanosis or respiratory difficulty. When the child was 2 or 3 months old the parents observed some inactivity of bodily segments and a tendency to keep the hands clenched with the arms flexed on the chest. At about the end of the first year, the mother noticed some body inactivity, especially of the left side of the body; the boy would slump somewhat to the left when propped against a pillow. Early in the period of development of the normal speech a form of mild dysarthria, or hesitation in speech, was noticed.

When the child was 5, i. e., two years previously, a definite change occurred. Without previous acute or insidious constitutional signs, attacks of disturbance of consciousness, associated with motor manifestations of almost constant pattern in the nature of petit mal began. An unusual feature was that the provocative factor was usually some sudden and unexpected affective stimulus. The attack was characterized by mild confusion or clouding of consciousness, with a spiral, rotary movement of the body and head; the body was turned in corkscrew fashion, the left arm was flexed and the right extended; the body usually turned to the left, and the head and eyes slowly followed; the pupils were momentarily dilated and fixed. Recovery was sudden and complete. The seizures varied in number, from a few to twenty or more daily. Fatigue had an influence on their recurrence. What is of interest is the gradually increasing influence of the patient's will in preventing or modifying the seizures. By a strong effort he is attaining greater mastery over them; he cannot induce them. There is no subsequent headache, confusion or somnolence, and no aura.

Neurologic Examination: The cranial nerves, including the fundi and fields, showed no pathologic change. The patient was right handed. Posture and gait revealed the absence of normal associated movements, more marked in the right upper extremity, and there was a suggestion of a flexor pattern in the latter. There was clumsiness in performance with the left arm and leg, emphasized in all coordinated skilled movements, as walking, skipping, pinning or buttoning garments, handling knife and fork, and in pointing tests. Rapid alternating movements were poorly performed on the left. Sudden arising from the floor on command resulted in a tendency to fall backward and somewhat to the left. Hyperextension of the left leg at the knee and a gradually increasing tendency to pes equinovarus were present. On the left side one suspected some degree of dyssynergia of the simple type, as revealed by a mild degree of Gordon Holmes noncheck phenomena; the left knee reflex approached the pendular swing. Muscle tone appeared normal. The dysarthria was of slight degree and was observed in early years. The reflexes, superficial and deep, were all present and were essentially equal, with perhaps a slight degree of greater response on the left, although the abdominal reflexes were also somewhat more active on the left. The plantar responses were flexor. The laboratory tests, including blood chemical determinations and cytologic tests and

studies of the spinal fluid were all normal. Stereoscopic examination of the skull did not reveal any abnormality. The intelligence quotient was 85.

Bárány Tests (by Dr. Almore): These did not show any evidence of abnormalities of the labyrinthine or cerebellar mechanisms. There was, however, a manifest loss of appreciation for the lower tones of the tuning fork, and vertigo was induced with difficulty. Pointing tests gave normal results.

Comment.—The diagnosis of the site of the pathologic condition may be narrowed to one of two locations: the cerebellum or one of its peduncles, or a more diffuse lesion of the extrapyramidal mechanism.

Tonic or cerebellar fits vary sufficiently in type to justify consideration of their presence. However, even in unilateral cerebellar fits, while the tonic spasms are more marked on the ipsilateral limbs, the opposite ones do not remain unaffected. The former become rigidly adducted to the trunk, while the contralateral are abducted. The spiral turn of the trunk and of the head and eyes is away from the side of the lesion, that is, toward the sound side.

The fragments of decerebrate rigidity, the loss of normal automatic associated movements of the right arm, the gradually increasing striatal pattern of the left lower limb, the defects, apparently of congenital origin, of the posture or pattern mechanism, lead one to regard abiotrophy or a developmentally defective extrapyramidal system as the basis for the symptoms in this case. It appears not improbable that, as a more intricate mechanism with its demands for function developed, a failure of the necessary tracts to appear may be the cause of the maladjustment, causing lower centers to remain uninhibited by a proper controlling mechanism. The influence of affective stimuli in "setting off" the seizures would suggest implication of the basal ganglia of the forebrain.

Medication did not have a favorable influence; phenobarbital has induced some somnolence and increased the coordination, without any material influence on the attacks.

DISCUSSION

DR. WALTER KRAUS: The attacks of petit mal mentioned seem similar to the static fits in epilepsy described by Hunt. This kind of seizure is probably closely related to what Wilson commented on in lenticular disease; that is, a definite degree of muscular weakness which is not accounted for by involvement of the pyramidal tract, usually believed to be the cause of muscular weakness. The uncertainty as to the site of origin of such weakness is emphasized by this presentation, in that Dr. Goodhart has not stated definitely whether the origin of the attacks of petit mal are in the midbrain, striatum or elsewhere.

DR. ABRAHAMSON: I have seen such attacks and such positions in postencephalitic patients, especially those with disturbance of the lenticular ganglia. It is difficult to call these attacks petit mal, or to label them at all. I think it best to call them postencephalitic or chronic encephalitic attacks. The slight cerebellar symptoms, I think, are secondary. The main brunt of the disease in this case is borne by the lenticular ganglia. There are many encephalitic patients, especially the very young, who show peculiar cephalogyric and oculogyric movements. The whole attitude of the body and the long course of the disease impress me as a postencephalitic condition. From the fragments of the history that I obtained, this condition apparently came on at a distinct period after birth.

DR. GOODHART: The attacks are only of two years' origin, but the condition was noticed first about two months after birth.

Dr. Jelliffe: I should like to ask about an evident thalamic over-response in the facial musculature, and about the distribution in the right arm and left leg. Does Dr. Goodhart postulate multiple lesions, or one? If the latter, where is it?

Dr. Goodhart: It seems to me that the overaffective unilateral reaction of the facial musculature would coincide with the condition which I mentioned as associated with thalamic influence: that surprise and any affect stimulus seem to be the inciting cause of these attacks.

Dr. Kraus' remarks are pertinent, in that, in spite of the fact that the pyramidal tract is not involved, there is a definite disturbance in voluntary control and in motor power; while it probably is due more or less to disturbances in coordination, one finds definite reduction in strength on the left side.

I am disinclined to accept Dr. Abrahamson's suggestion of postencephalitis as the etiology on account of the early beginning of the motor disturbance and its insidious onset. Whether something else has occurred to bring on these attacks of petit mal of the past two years I do not know, but to me it seemed more likely that the condition is an abiotrophy or lack of development of the extrapyramidal system.

Thymergastic Reactions and a Consideration of Certain Types of Hypochondriacal Hypothymergasias (Hypochondriacal Depressions). Dr. Leslie B. Hohman (by invitation).

[Only the conclusions of the paper as read are given here. The full paper will be published at an early date.]

I shall emphasize: (1) The value of a thoroughgoing experimental empiricism in psychobiology and psychopathology — an empiricism which elevates psychiatry to a real scientific discipline in that it arranges all cogent facts in sequence, and exacts of these factual sequences only two rules: (*a*) that they will regularly lead to the same outcome; (*b*) that these sequences prove their cogency by possessing predictability or experimental reproduction. This position refuses to deny the autonomy of the facts of psychobiology, because they are not reducible to the facts of the physical sciences. (2) An experimental attitude and approach to the study of normal and pathologic emotional states, as well as the need for studying these reactions as total organism responses to the situation. (3) A group of cases showing marked hypochondriasis as the main symptoms, which are fundamentally emotional reactions of circumscribed duration; the clinical recognition of these states comes from a study of the "mechanics" of the reaction or behavior pattern. (4) To represent this objective or experimental empiricism in psychobiology and the need for accepting all cogent facts in the study of the emotional reaction, I propose the adoption of the meyerian term, thymergastic reactions, for the general group of pathologic emotional disturbances.

The Value of Combining Methods of Treatment in the Psychoneuroses. Dr. Thomas W. Salmon.

Attention is called to the desirability of combining the three methods of healing in the use of psychotherapy which would lead to an increase in the success of this method of treatment. Another enormous advantage would be that the psychoses, diseases which are avoided by the three great psychotherapeutists advocating the three methods, might sometimes be benefited by their ministrations. There are advantages, however, much wider than these,

advantages to those who are engaged in the work itself. Unusual opportunities are opening before the psychiatrist in the schools, prisons and colleges. If he fails in the prison and the children's court and in the college to modify the thinking, feeling and conduct of the people brought to his attention, he should abandon the field to some one else. It is in this new field of effort in psychiatry that the combination of the methods of treatment is to be most required. One of the best illustrations is to be found in the training of children. A special technic must be evolved. Before this combination of methods can be effected, there must be facilities for joint efforts. The patients are dealt with in series at the clinics, and afterward, if this is followed by a conference, there is an opportunity for the combination of methods. No psychiatric clinic is efficient or complete unless it has a place for the employment of the main methods of psychotherapy. In addition to that are needed more tolerance, greater frankness as to negative results, and the adoption of a broad spirit of purpose, rather than the carping, critical attitude of a special point of view.

DISCUSSION

DR. GOODHART: Among the many points suggesting discussion in these papers is one of interest to neurologists and psychiatrists; namely, the nomenclature and attempt at classification suggested in the title of Dr. Hohman's paper. The term, thymergasia, as I recall, was introduced by Dr. Meyer. Before we can understand each other, we must speak a common language. Neuropsychiatry has long been suffering from just this lack of consistency in terminology; every now and then new terms are introduced or are variously and loosely used. Neurology is undergoing a thorough housecleaning in that respect, and the work of a joint committee establishing a rational nomenclature will soon be presented. The same should be done in psychiatry. The question of etiology, the therapeutics and prognosis should not rest on such a classification as that which is represented by the affective psychoses — manic-depressive or so-called benign psychoses as distinguished from the more malignant as represented by dementia praecox. A much more rational and comprehensive basis would be rather an analysis of the life reaction of the individual; I have no doubt that all see schizophrenic thinking often in the so-called functional cases — the neuroses and psychoneuroses — and certainly in the frank depression psychoses. Dr. Hohman has broadly intimated this as a principle in his analysis of the psychoneuroses. It is only by a study of the life reaction of the patient that one may form some idea of the outcome. By the life situation of the individual, I mean what he has received from his progenitors, what his own life experience has been, what has happened to him in the course of his acute illness and its immediate exciting cause, and, of no small importance, what influence will his recovery have, or does he think it will have, on his future life. As Jung has pointed out, the psychosis, and I think one might also say the psychoneurosis, may be the saving, the beneficent turn in the patient's life; it may place him where he belongs, indeed it may be the fulfilment of a long suppressed subconscious desire and give opportunity for the expression of latent potentiality. Indeed, only through this psychopathologic experience may the individual really "find" himself. In the early years of my practice, a most exemplary demonstration was brought under my observation. To a perplexing and profound problem concerning his future, a minister in a small town reacted by a complete amnesia; he might just as well have developed a psychoneurosis or, indeed, a psychosis. Instead, however, his conscious life was pushed in its entirety into the sub-

conscious. The new personality emerging from the subconscious revealed the suppressed hopes, ambitions, desires, moral and strictly intellectual characteristics of a personality different from the former active personality. A study of the new personality enabled recasting and readjustment, so that an entirely new life activity was furnished. The patient was freed from the limiting bonds of clerical activity, and a new field of endeavor was given him. I am satisfied that the patient was on the brink of a mental dethronement and that the pathologic reaction established him for his life work.

Dr. Salmon has made a most commendable plea for a unity of purpose and has with characteristic clearness shown us the way.

Dr. Brill: Dr. Salmon views the situation as it really is. There is no doubt that such a state of affairs exists; the various schools are at war with one another. But Hobbs, the philosopher, made this clear long ago when he said that we are all at war with one another; yet it does sound surprising to hear that psychotherapists do not behave differently. Dr. Salmon himself is not against controversy; he thinks that it sharpens the mind and I agree with him, and still I feel that he is perfectly right in deploring the fact that there is no cooperation among schools that strive to accomplish the same purpose. Freud himself said long ago that every physician practices psychotherapy whether he knows it or not; I know that he always took a broad-minded view of the situation and never displayed objection to other methods. It is the other schools who always voiced objections to his methods. I practiced a great deal of hypnotism, persuasion and reeducation long before I heard of Freud, but I found later that psychoanalysis gave me more understanding than the other methods. Freud shows the actual dynamics at the basis of symptoms. I believe that psychoanalysists were really the first behaviorists, with all due respect to Dr. Watson, for psychoanalysis teaches that no two cases are alike and that every patient has its own individuality. That is the reason why I prefer Freud's method of psychotherapy, but I see no objection to any other school. As a matter of fact, not every case is accessible to psychoanalysis, but personally I feel that even in such cases the psychoanalytic approach is by far the best.

Dr. Hohman's presentation reminded me of an Italian proverb "Si non é vero é ben trovato," which means that even if it is not true, it sounds well. Dr. Hohman described man as a fine, working piece of machinery to which one can add this device or that device, and can keep on modifying and changing these devices ad infinitum. The only difficulty presented in such a concept is that man does not act in this way. He is adaptable and flexible, and if anything happens to disturb him, he does not always react in a definitely expected manner. I could match Dr. Hohman's ten cases with ten equally well worked out and give my own interpretation, and one would surely find the differences of opinion that Dr. Salmon has so earnestly deplored. Everything Dr. Hohman told is undoubtedly true according to his view of his cases, but I feel that his ideas are too mechanical. Enough is not known about the human emotions to speak in terms as definitely as he presented. What he says about the psychoanalyst's interpretation of the psychoses is not quite true. There are many psychoanalysts, just as there are many psychotherapists, and not all of them think that just because a certain psychosis does not react to psychoanalytic treatment there must be an organic lesion. One does not know this. Some believe it to be so. Bleuler takes this view; but Bleuler, the psychiatrist, although feeling that without Freud there would be no psychopathology, has his own ideas about the structure of certain psychoses.

I agree with Dr. Salmon that for some people it is not necessary to take everything that Freud offers. One is privileged to take what he wants; one takes what one can digest. Some follow Freud in all cases, I do this because I find that I can harmonize his theories of the psychoneurotic person and the psychosis with my views of life, that is, with the views of life I have gathered by studying normal and abnormal people. I can understand my cases when I look at them in the light of psychoanalysis, whereas the other methods do not offer me the same opportunity.

I would like to discuss some of the other points made by Dr. Hohman, but I hesitate to take them up lest I should make some mistake. I would prefer to read the paper first and then discuss it. However, I feel that Dr. Hohman's paper is a distinct contribution. I cannot accept his classification. I believe that psychiatry now has a fairly good working classification in Kretschmer's cyclothymic and schizothymic or in Bleuler's modifications of Kretschmer. The cases which Dr. Hohman cited as manic I would say were schizoid, or preponderatingly schizoid. Some probably belong to Bleuler's schizoid manic type. I cannot agree with Dr. Hohman that heredity and constitution count for nothing, much as I believe in environmental influences. I have observed a number of families in which the parents were both markedly burdened by heredity, schizoid and syntonic, respectively. In one of the families there are four children, the oldest of whom I have known for sixteen years. This boy was distinctly manic in make-up and continued to show a syntonic behavior until he was about 14 years old, when he suddenly changed into a schizoid manic. The second child is distinctly schizoid, and now, at 14 years, she is very schizoid in make-up. The third is again very manic, while the fourth, a boy of 8 years, is also manic. I have closely observed these children with the parents, who are intelligently interested in the problem, and I have no doubt about the constitutional factors. There is no question that there is such a personality as Kretschmer's schizoid type and that this personality reacts in a schizoid way throughout life, regardless of environment. The same may be said of the syntonic types but from schizoid *A*, who is an adjusted personality and who accomplishes a great deal in life, to the schizophrenic *A* there are a great many transitions.

Dr. John B. Watson: I wish to congratulate Dr. Hohman on a clear and brilliant paper. I was interested to hear him take as conservative a position as he did on the question of the fundamental constitution of the individual. I know he took the only scientific position that is possible, but I do want to say that the behaviorist is after this constitutional factor. I think he has gone far enough in his experiments and conclusions to deny the existence of such a factor. I would like to see one good case shown by the biologists or the psychoanalysts or the psychiatrists in which the facts cannot be explained on some basis other than that of the so-called constitutional factor. It is perfectly possible to take infants who have been brought up to behave in one way and after six weeks train them to behave in another way. When I speak of children, I mean children too young to go into the hands of a psychoanalyst, and in general too young to go to the psychiatric clinics. I refer to children 1½, 2, or 2½ years old. One can almost make and remake them at will, and in a few weeks time. Proof is not definite, but I should like to go on record that the behaviorists can get along without assuming any mystical constitutional factor. I believe that man is made, not born.

Dr. George Draper: I am interested in the work of Dr. Watson and am glad to see that he bears all the earmarks of having descended, like the rest

of us, from preceding generations. I must say that it has been difficult for me as an internist, quite out of place here, to escape the belief that there are inherent susceptibilities in man which render him a specific reagent in disease-producing mechanisms. Now I cannot see why there is any great difference between the psyche of a human being and the liver, for example. Certainly people who develop pernicious anemia are peculiar and different, not only in their morphology but in their psychology and in various aspects of their metabolism from other people. The recent work on the liver in relation to people having pernicious anemia, it seems to me, is opening a vista of the most astonishing sort, pointing directly to a lack in their physical organization of some substance which is essential to normal development of the blood-forming organs. It is a little difficult, in view of an example of this sort, to rule out, at least from the physical body, the question of forces inherent in heredity. Certainly the racial distribution of disease is also suggestive. Here again the pernicious anemia is illuminating, as one knows that pernicious anemia is almost entirely limited to the northern peoples. It is curious that the livers of people of these races seem to lack something that the livers of the more pigmented peoples possess. It is hard to think of man except as a total organism which might be spoken of as a "mind body." Certainly if there is no doubt of the inheritance of physical and physiologic characters, it is logical to anticipate some hereditary influence which is expressed in characters of psychic pattern.

Dr. Foster Kennedy: Sir Thomas Browne, who had more wisdom than we have knowledge, warned us not to be proud that we have dignity, modesty and contentment, for all of these lay in the egg, before we were. So Dr. Watson disbelieves in heredity and feels that a human sperm and ovum joining anywhere else than in the uterus would not necessarily produce a human animal. Did Dr. Watson ever hear of an experiment of the Glasgow County Council, about twenty-five years ago, who decided to take the slum foundlings of Glasgow, previously kept in foundling hospitals, and farm them out in the hills and islands of Scotland? These children were waifs from the Glasgow slums, which are among the worst in the world, and their stock was of the poorest. They were put in as good an environment as any child could be brought up in, on small farms of "four acres and a cow" with fine peasants as foster parents, and in fifteen years they had polluted the countryside.

Dr. Watson: I should like to ask Dr. Draper if he is familiar with Child's work on the effect of temperature, pressure, etc., on the developing organism? Whether he thinks that if the egg and the sperm, after being united, grew up in any other place except in utero, there would develop such a thing as an arm, or a leg, or even a liver? I think it is extremely doubtful whether there would be anything that would take on the semblance of a human being. The conception of heredity will have to be renewed — even of the inheritance of bodily shape, form and size.

An experiment is being made in Germany which I have not seen yet. Children from poor stock were taken away from their homes and reared and cultured under the best behavioristic conditions — as far as they have got to that in Germany at the present time. The results, I hear, are so satisfactory that the officials in charge are suppressing the facts for fear they may injure the home.

Dr. Jelliffe: Catholicity is to be practiced as well as preached, and there is no necessity for any doctor to call a man a "quininologist" because he happens to use quinine for malaria, or to call him an "arsenicist" because he uses arsenic for

anemia. There is no necessity for calling a man a "psychoanalyst" because he might analyze this patient, or a hypnotist because he hypnotized that one, or a pedagog because he may attempt to reeducate a third. Man does not need labels like canned goods on the grocer's shelves. So far as what Dr. Salmon tells us, I am in absolute agreement. More catholicity, charity and kindness, and more of what Dr. Kennedy has reminded us of in his quotation from Sir Thomas Browne are needed.

Coming more specifically to Dr. Hohman's contribution, it seems to me that it is undesirable in a way to make these antitheses so striking; antitheses between constitutional and dispositional factors, except as it may be advisable to point out wherein an unsuccessful approach from the constitutional side or the same from the dispositional side may lead into serious breaches of that balancing of judgment which we have been told about. There are a number of points Dr. Hohman discussed, which are due to the persistence of this tendency to destroy the other side. From the standpoint of logical determinism, I cannot see wherein (in the state of the paranoid trend of my psychosis) one can avoid applying the strictest logical criteria to the working out of a hypothesis, and I cannot understand Dr. Hohman's objection to hypotheses. Everyone uses hypotheses. They begin as fictions, as Vaihinger has reminded us. I have spoken frequently here of the value of the suppositional point of view, as of regarding practically everything in logical terms as pure fictions. If the fictions work, then one may build up hypotheses. If the hypotheses work, then one may build theories, and if the theories work, then one may build laws. A series of logical steps results, which it seems to me Dr. Hohman has in a certain sense overlooked, because he has denied the felicities of classification, and yet he has told about "groups" and certain patients that were "allied"; thus, in spite of himself, he is possibly committing the logical fallacy that he is deploring in his paper. I am in sympathy with him that the accent has been far too strong on the constitutional and hereditary factors in the attempts to understand disease. I agree with him that such attitudes of mind constitute serious barriers to the kind of empiric study that he is contemplating. Every tub stands on its own bottom. Each case has its own pathology; therefore one has to be empiric; on the other hand, there is the necessity of grouping, and if it were not for the faculty of making what are called classifications, why there is a great deal of wind-jamming done, which I fear I am doing myself, in diffusing a situation so widely that it is impossible to bring it together. I shall close with just a reminiscence, since a little Latin and a little Italian have been quoted and I am not going to give the original Greek, but I will remind you of Plato's discussion with Protagoras, in which one sees the two attitudes of mind which Dr. Hohman has nicely portrayed; the mind of Protagoras, the empiricist, and of Plato, the absolutist. Protagoras insisted that man was the measure of all things — each saw as he was made. The Platonists had their absolutes, as their modern followers have their "disease entities"; they fix names as things — with Dr. Hohman, I am on the side of Protagoras.

Dr. Monroe Meyer: While Dr. Hohman presents himself as the representative of a model empiric scientific discipline, it seems to me that he betrays, at the same time, a certain degree of lack of knowledge of historic facts concerning a scientific method that he is disposed to criticize. Dr. Hohman appears inclined to imply, if not directly to state, that psychoanalysis is essentially a dogma. Further, he evidently feels somewhat disparagingly toward what psychoanalysts have contributed to the solution of the problem of the psychoses.

Psychoanalysis has been constantly modified by Freud and his school as new observation and discovery permitted. Twice in the history of psychoanalysis, Freud literally tore up his own theories with his own hands. I refer to the theory of infantile sexual trauma and to the theory of libido-anxiety conversion. Freud did not hesitate to subject both these theories to radical revision when fresh facts so necessitated, an attitude which is anything but dogmatic. As regards psychoanalysis and psychiatry, I refer Dr. Hohman to Dr. John Rickman's excellent survey of the subject in the last number of the *British Journal of Medical Psychology*, a perusal of which will, I believe, leave him willing to concede that psychoanalysts have, perhaps, after all, thrown a little light on the issues obtaining in some of the psychoses.

Dr. Oberndorf: Dr. Salmon's paper emphasizes the coolness which the various schools of psychiatric approach formerly assumed to the revolutionary teachings of Freud, and yet I think that Dr. Salmon himself might be willing to admit that both the suggestor and the reeducator of today are largely indebted for a more rational and effective application of their methods to the psychologic mechanisms developed by Freud and employed by them more or less constantly, consciously or unconsciously. Dr. Hohman himself, notwithstanding his opposition to the psychologic approach, uses the freudian nomenclature in his paper frequently. The main difficulty of combining methods of treatment in psychoneurotic conditions, as suggested by Dr. Salmon, rests in the vitally different aspects of approach by the three groups of psychotherapists mentioned by him. The analyst with his primary aim of having the patient do most of the work cannot successfully depart too far from that position. I agree with Dr. Salmon that it is desirable to avail oneself of all three methods, and others as well, but they cannot be combined by one man effectively. The greatest advance will come when one is able to determine more accurately than today just what type of case is most suitable for the psychoanalyst, and what is best referred for treatment by other methods. It is distinctly disadvantageous to treat one patient by a combination of methods, but it is preferable to refer a particular case to a particular expert in the type of therapy best suited to the individual and his disease.

Dr. Hohman: I am sorry to be the cause of so much discussion, and I cannot possibly answer all the criticisms, but I think there is one thing that is the crux of the matter. Dr. Jelliffe says I object to classification. Nothing could be further from my thoughts. There must be classification; it serves an important purpose, and I should be sorry for myself if I were not able to see similarities between cases of similar sorts. It is a classification that has as its presupposition the existence of a disease entity, that is a specific pathologic process, to which I object. I described a group of cases that had certain common features of reactivity, and these I called hypochondriac reactions. My objection is not to calling them by a name, but to assuming that a special disease process lies behind them. The question of how emotional reactions can come, without a constitutional factor, to be what is finally called manic-depressive insanity is a thing about which I wished to speak. To illustrate the fixity of psychologic patterns, I bring up the question of language mechanism. The particular language that an individual speaks is wholly due to environmental factors. Whether I speak German or English is purely dependent on which environment I grew up in, and that particular language becomes so structuralized in myself that if I tried to shelve that language after my twelfth and certainly by the twentieth year, I believe that I should never eradicate the original mechanism laid down in the first years of life. One

can never eradicate the traces of the original language. That is, a pattern conditioned from the outside, is woven into the structure of the individual. That is the type of fixity of psychologic patterns which I mean. I quoted experiments of Dr. Watson which show easily that one can build up and tear down emotional patterns. It seems to me from the therapeutic point of view that one should realize the things that are modifiable, especially since experimental evidence is forthcoming that one can alter the very patterns which look as if they might be hereditary.

PHYSICAL CONSTITUTION AND GENERAL PARALYSIS. H. A. BUNKER, JR., M.D.

The study represents an attempt to approach, from the standpoint of the morphology or physical constitution of the patient, the problem of the *raison d'être* of general paralysis. The latter is a problem of some obtrusiveness for the reason that a situation wherein a small minority of syphilitic individuals fall victims to neurosyphilitic disease of the parenchymatous type, while the overwhelming majority of syphilitic persons escape, necessarily challenges inquiry into what it is that underlies this notable difference in the reaction on the part of different individuals to syphilitic infection.

Two observations are of primary importance in relation to the natural history of general paralysis. One of them is that during the first months of syphilitic infection invasion of the central nervous system, as measured by the presence of abnormality of the spinal fluid, takes place in at least one third of all patients, while in a small minority of these — perhaps about 5 per cent — the changes in the spinal fluid are intense and persistent, and are resistant to antisyphilitic therapy. It is possibly from among the latter individuals that the future general paralytic and tabetic patients are recruited. The second observation, for which Joseph Earle Moore is responsible, is that, whereas in early syphilis the incidence of abnormality of the spinal fluid is the same in women and in men and in late syphilis the same is true concerning men and nulliparous women, the incidence of abnormality of the spinal fluid is definitely lower in late syphilis among women who have had several pregnancies subsequent to infection; this seems to indicate that pregnancy, and especially the occurrence of multiple pregnancies, conferred a material degree of protection against serious syphilitic involvement of the nervous system.

Since Dr. Draper, among others, has demonstrated in various instances that a predisposition to certain diseases may be reflected in the morphologic or anatomic characteristics of the subject, it seemed reasonable to ask if a general paralytic patient is by any chance a different kind of individual from one not having general paralysis, whether this difference might similarly be reflected in the physical make-up of the two sorts of individuals. My associates and I have therefore carried out, on a series of 100 cases of general paralysis in men and on a group of sixty-four supposedly normal men intended to represent a random sample of the general population, a series of standard anthropologic measurements consisting altogether, in accordance with the technic employed by Dr. Draper, of about forty-nine body measurements, together with forty-four indexes or ratios calculated from various pairs of these measurements.

Classifying the material in the manner of Kretschmer, I found that 69 per cent were of the so-called linear type of physical habitus (asthenic, asthenic-athletic, or athletic) and 25 per cent of the lateral type (pyknoid and pyknotic). This is in agreement with the observations of Gründler, published since the present study was undertaken; 69 per cent of his eighty cases of general paralysis in men and women were of the linear type and 20 per cent of the

lateral type. This observation is in definite contrast with the normal control group of 118 cases reported by Gruhle, of which only 42 per cent were of the linear and 34 per cent were of the lateral type.

With regard to the results of the anthropometric part of this study, I believe that a certain number of anatomic differences, which appear to be significant, between general paralytic and nonparalytic patients have been worked out; i. e., they appear not to be merely chance differences. It would seem, then, that syphilitic persons who develop general paralysis are in some way a different kind of people from syphilitic persons who do not; and what is here noteworthy is that the difference between them should be reflected in a number of anatomic features, some of which give expression to this underlying difference in a higher degree than do others.

DISCUSSION

Dr. Draper: There is no question that the general paralytic patient is different from the tabetic patient, and certainly he is different from members of other disease groups. I am not sure whether one can safely use a so-called normal control group because of unexpressed disease potentialities. One may, for example, belong to a given disease type and may never develop the disease, because he chances to escape the particular menace to which he is susceptible. There are, however, many other things about the general paralytic patient which cannot be measured, but which differentiates him distinctly. About sixty years ago, Nacke made a morphologic study of general paralytic and tabetic persons and brought forth evidence to show that the general paralytic belongs to what is now commonly known as the syntonic or pyknotic type. Consequently, I was surprised to find that this group was largely of the asthenic type. Notwithstanding these conflicting observations, there is no doubt that persons with neurosyphilis are different from those who have vascular syphilis, for example. Neurosyphilitic persons are of the hairless type and have a smooth skin and rounded contours, while most of the general paralytic patients I have seen are of the thick-set type. Dr. Bunker's observation on this point is interesting. In the study of constitutions, it seems to me that dependence on mensuration alone is not wise. Emphasis on pure morphology, particularly osteology, is not sufficient. Nevertheless, there are certain obvious morphologic characteristics in general paralytic patients which make it clear that the subjects of general paralysis are different from those who develop other forms of syphilitic disease.

Dr. Hyslop: It was four years ago that Thomas K. Davis published statistics bearing on the constitution of the general paralytic person. His conclusions were to the effect that general paralysis is more common in the pyknotic type, and if it occurred in the asthenic type, it ran a more benign course than in the asthenic type. I wonder if neurologists have found reason to refute this tentative conclusion of Davis, and whether in their experience general paralysis does run a different course in the asthenic type than in the pyknotic individual.

Dr. Jelliffe: It happens that I have been thumbing over rather rapidly the ninth edition of Kraepelin's second volume of Clinical Psychiatry, which has just appeared. You know with what care Kraepelin had been working on that particular aspect of the problem. Those who came into contact with him during his trip to America know how keen he was in testing out the hypothesis of Doraschiewicz, i. e., that people who have been vaccinated were those who develop general paralysis. They conceived that there was some intimate relationship between skin metabolism and brain metabolism, which

seemed more or less likely. There might be some relationship between the skin and susceptibility and immunology of the skin and the capacity for involvement of the nervous system and more particularly of the parenchyma of the cortex. Both Kraepelin and Plaut came to negative conclusions and could not establish any relationship between vaccination and the possibility of general paralysis. I want to ask Dr. Bunker, if I heard him aright, when he drew a certain inference about the comparative proportion of the occurrence of general paralysis in women and in men, whether he took into account the comparative amount of syphilitic infections in women and men.

Can increase in the length of the proximal bony structure be correlated with muscular situations? In response to certain muscular activities a reaction in the bony structures occurs, and on the basis of some unknown unconscious factors with reference to the utilization of the strong muscles, as it were, of the trunk, might there be some relationship between muscular libido and change in bony structure?

What is the susceptibility for the cortical structures to be involved? It has frequently been said that the people who do not use their brains, as the nomadic Arabs for instance, do not have general paralysis. This general notion lies at the bottom of the "syphilization and civilization" slogan, popular for many years. It has remained for many careful studies to show how false such a generalization was. At any rate, the whole problem sums itself up in the fact that there are differences in constitution or disposition that determine the general paralytic reaction. To determine what these differences may be is a research problem. Possibly it lies in an interrelationship between constitutional and dispositional factors. The only contribution I can make to this subject is one I made many years ago. It dealt with the problem of localization of the syphilitic process in the central nervous system on the basis of certain dispositional factors. It concerned the localization of crises in tabes. There are gastric crises, laryngeal crises and rectal crises, and other definite localizations of the tabetic process in certain parts of the nervous system. The observation I offered concerned a localization factor of crisis. In three cases of rectal crises, I had the opportunity to study with a considerable degree of persistence and depth the localization of the crisis which was possibly determined by definite sodomistic fixations on the part of the individuals. This sodomistic fixation may have had something to do with the tensions in the autonomic segment and thus have determined the breakdown at that particular segment. Whether one can speak of the brain as an organ I do not know. One knows that the brain is a mosaic of organs and that, as yet, complete cyto-architectonic studies have not been made in general paralysis, so that accurate localization of the involvement is still unknown. There have been no cytotectonic studies that in any sense of the word faithfully supply such criteria as the work of von Economo demands. Until such studies have been made, not enough is known about the distribution of the lesions in general paralysis, and therefore they cannot be correlated with matters of organic function.

DR. JOSEPH SMITH: It is difficult to see how studies of this character can elucidate in any definite way the relations between specific infection and possibilities of later acquiring general paralysis. It would seem that such items as the length or circumference of the long bones can have no bearing, either on the acquisition of a specific infection or on the localization of the virus, or can be a determinant of conditions which in one case will result in constitutional syphilis and in another will affect the parenchymatous structures of the brain. Moreover, if there were a possibility of showing the existence of such

relationship, it would not tend to clarify matters but would rather confuse the problem, for the question would only be shifted and another raised: "What is the intrinsic worth of such relationship?" As to the statement that about 4 per cent of those who are infected with syphilis later acquire general paralysis, I should like to add that general paralysis is primarily a mental disorder, though accompanied by physical signs and having an organic substratum. In any community, there is a certain ratio of the insane population to the mentally healthy, and one can easily conceive that among syphilitic persons there is a corresponding ratio between those who acquire general paralysis and those who escape it.

DR. BUNKER: There is a large element of subjectivity involved in classifying patients according to the Kretschmer types — so much so that when two German psychiatrists in the same clinic went over a women's ward in order to make a census of this character, one of them found almost twice as many pyknotic patients as the other; the close working relationship of the two men did not obviate a vast discrepancy in their results in deciding who were and who were not individuals of the pyknotic type of physical habitus. I have tried to make my classification as objective as possible by having another opinion on the same cases and by supplementing the observations with the so-called morphologic index suggested by Wertheimer.

As to Dr. Hyslop's question, I am under the disadvantage that these were patients who had been subjected to treatment by malaria or by tryparsamide, and therefore the therapeutic outcome is hardly to be compared with what I understand Davis reported. The two series of cases are not comparable, though there would still remain the problem, of which Dr. Hyslop has spoken, of tracing the correlation between the type of individual and the response to malaria or other treatment.

Dr. Jelliffe raised a point to which I laid myself open, namely, the relationship between males and females with reference to the acquisition of syphilis. In the case of general paralysis, with the ratio of 4:1, I took the patients admitted in the last fifteen years at the Manhattan State Hospital, and Dr. Jelliffe's criticism undoubtedly applies. Other clinics have reported higher ratios, and Gärtner says that an allowance must be made for the difference in the existence of syphilis in males and females, but how much he was not able to say, and neither am I. Moore, however, has, as I recall, reported an equal incidence in males and females in all syphilitic groups except primary syphilis, latent syphilis, and neurosyphilis.

Regarding the proximal bones of the two extremities, what my figures have brought out so far is not any absolute difference in these, but a relative difference in the sense of the distal bone being long in proportion to the entire extremity in question, as compared to the control group. What that may mean I have no idea. This is only intended to be a provisional report, and I hope to work out the data much more fully, so that it may be possible to express some of these relationships in perhaps a more convincing form with regard to the predictional chances of any syphilitic patient becoming paralytic or not, depending on his exhibiting one or more of the entire number of characteristics by which it is shown that general paralytic patients differ from normal ones.

I can only say that undoubtedly general paralytic patients are different from normal people, and the remarkable fact, if it proves to be a fact, seems to be that these differences should be exhibited anatomically as well as in other more complex and obscure ways, into which one has so far scarcely penetrated.

SOCIÉTÉ DE NEUROLOGIE

PROF. G. ROUSSY, *President, in the Chair*

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AMYOTROPHIC LATERAL SCLEROSIS IN A SYPHILITIC PERSON. By DRS. BABONNEIX and WIDIEZ.

A mechanic, aged 44, complained of the gradual onset of paralysis with atrophy and involuntary muscular contractions in the upper limbs, of about sixteen months' duration. The picture presented was: atrophy of the upper extremities; increased reflexes in the lower extremities; slight deviation of the tongue and monotonous speech. The blood Wassermann reaction was positive; the spinal fluid was entirely normal. Since the pupils responded normally and the spinal fluid reactions were negative, neurosyphilis did not seem to enter into the picture. Although some have believed that spinal syphilis may take this form, the subject is open to question.

DISCUSSION

DR. LÉRI: There is no reason to believe that Charcot's disease is syphilitic; it appears to be autonomic; even its course is not that of syphilitic infection. Yet, cases are not rare in which a syphilitic lesion in the anterior horns and lateral columns may simulate this disease more or less completely. In these cases the amyotrophy precedes the signs of spasticity, and the evolution of the condition is infinitely slower. Such are the cases of medullary syphilis simulating amyotrophic lateral sclerosis which I have described.

UNILATERAL NYSTAGMUS OF THE PALATE AND RESPIRATORY DISORDERS IN PSEUDOBULBAR PALSY. DRS. GUSTAVE ROUSSY, GABRIELLE LEVY and NICOLAS KYRIACO.

The patient, aged 42, came under observation for left hemiplegia, with disorders of speech and deglutition and spasmodic laughing, which began, in 1917, with a stroke and was followed by a second attack, in 1921. This had affected the right side and had left her speechless for a while.

On examination, the left hemiplegia was predominant, the speech was nasal and articulation was defective, with difficulty in enunciating labial and nasal sounds. Besides this, the voice was dead, and breathing was quick and short with no respiratory pause. The patient said she became out of breath when making a long sentence. There was spasmodic laughing, which interrupted speech now and then. Movements of the face and lips were carried out well. The tongue was protruded in the midline without tremor. The uvula deviated to the left and was the seat of myoclonus, arrhythmic but synchronous with movements of the left posterior pillar of the palate and the posterior wall of the pharynx. At each contraction the posterior pillar was drawn toward the median line, whereas the posterior wall of the pharynx described a curtain movement toward the left. These tics, which averaged from 90 to 100 per minute, were of unequal amplitude and sometimes notched. No other clonus was observed, even in the diaphragm, and the reflex of palate and pharynx was abolished. Stroking the pharynx did not produce nausea. Mastication was slow and difficult, and the patient coughed and choked after swallowing. Sometimes fluids ran back through the nose. Laryngoscopic examination showed no myoclonia, some weakness in adduction, and nausea could be excited by pressing on the epiglottis. Respirations were increased

in rapidity and the expiratory pause was lacking, but the diaphragmatic contractions were normal. The rest of the examination showed characteristic symptoms of mild hemiplegia on the left and traces on the right side, but without aphasia.

Although these palatal and pharyngeal myoclonias have been recognized for many years, the lesion responsible was recognized only recently by Foix and his pupils. In this case, the strict unilaterality of the jerks and the limitation to the palate and pharynx were remarkable; it is difficult to explain the absence of more marked respiratory disturbances. The pathologic lesion in such cases appears to lie in the central bundle of the tegmentum.

TWO CASES OF SYPHILITIC RADICULITIS OF THE TRIGEMINUS. ANDRÉ THOMAS and J. JUMENTIÉ.

CASE 1.—A woman, aged 47, suffered from frontal headache, and a month after the onset fell from a step ladder, striking the face in the malar region. Two days later she had diplopia, and for a week the left eye showed internal strabismus. She suffered stabbing pains below the eye and pricking sensations in the interior and also in the internal part of the nose, extending to the malar region; these pains began at the time of the accident. The skin over the nose and forehead became insensitive. Examination revealed a trigeminal lesion, affecting both motor and sensory fibers, although sparing the third sensory division. On the left side of the face there was almost complete anesthesia to pain and temperature in most of the territory innervated by the upper branch. The supra-orbital region and the nose presented only mild hypesthesia. There was hypesthesia in the distribution of the second division, only the upper lip being anesthetic. The upper part of the auriculotemporal field was hypesthetic, but the rest of the third division was intact. The cornea was insensitive, and the reflex was absent. Pressure on the foramina was painful. Six months after the onset, the whole side of the face had become anesthetic without noticeable differences, even traction of the hair on the left side being painless. Deep pressure sense was absent over the forehead and was diminished in the cheek. The sensibility of the mucous membrane was much diminished; the anterior half of the tongue had slightly reduced sensibility. The patient had difficulty in opening the mouth, and the masseters were evidently atrophied; they were painful on pressure. There were no trophic disorders of the skin or mucosa, and the sympathetic tests revealed normal observations. Lumbar puncture showed 230 cells per cubic millimeter, mostly lymphocytes, and reduced sugar and chlorides. The blood Wassermann reaction was positive. Arsenical treatment caused the headaches to disappear, and the pains in the face were considerably reduced. At the time of the report, the objective disturbances were disappearing. No other lesion of the cranial nerves and no evidence of central nervous system degeneration were found.

CASE 2.—A woman, aged 36, suddenly experienced pains in the right temporal region, extending over into the supra-orbital area, then to the back of the head, and finally to the neck. They were sharp, sticking and pounding, occurring for hours at a stretch with short intermissions of five or ten minutes. Sometimes they reached the cheek, sometimes the ear. There were no pains in the eye, although the eye felt full. Recently, the whole side of the face had become painful, and insomnia had resulted. In January, 1927, six weeks after the onset of the disease, the patient noticed that she could not feel with the right corner of the mouth. Vomiting once resulted from the severe pain. There was complete anesthesia for both pain and temperature in the cutaneous field of the right trigeminus. Touch was felt partially in the right side. The mucous membranes were also anesthetic and felt swollen to the patient; the nasolachrymal reflex was

absent on the right. Anesthesia to pressure was complete in the first and second divisions, and partial in the third. The tuning fork was poorly perceived. The masseter and temporal muscles were painful to pressure and atrophic, the jaw deviating on opening. Opening the mouth was painful, and there was reaction of degeneration in these muscles. There was no difference in color or moisture in the face, and the reaction to pilocarpine was equal on the two sides. Sinapisation caused equal flushing, and the pilomotor reflex was normal on the two sides. The palate contracted normally. Speech was somewhat hampered. There was slight nystagmic twitching but nothing else in the ocular examination. Hearing was perfect, and the labyrinthine reactions were normal. The cerebrospinal fluid showed signs of meningeal reactions, with 142 cells per cubic millimeter, a negative Wassermann reaction, and a colloidal benzoin reaction with precipitation in the meningitic but not in the syphilitic zone. The blood Wassermann reaction, however, was strongly positive. There was no disturbance in the trunk or limbs.

The similarity of the two cases is interesting, and there are certain clinical characteristics of these trigeminal lesions which allow one to indicate the seat and the nature. The pains at the onset were not exactly those of essential trifacial neuralgia, and were associated with pains outside of the trigeminal distribution. The nocturnal headache is also against trigeminal neuralgia. The appearance of objective sensory disorders in the region in which the pain began is a stronger point of differentiation. The topography of these sensory disorders does not conform to a peripheral, nor even to a ganglionic, distribution, but to the radicular fibers. The loss of deep sensibility is particularly important. The integrity of the sympathetic fibers is a most important observation, for it allows one to localize the lesion behind the point where these fibers join the trigeminus. All this, therefore, points to a retrogasserian lesion. Such lesions are not rare, because sources of irritation are frequent at the base of the brain; nevertheless, in these two instances, they are exceptionally isolated. Camus has reported a similar case, and another one of more extensive character also cleared up under specific treatment. Trigeminal lesions in tabes are well known, and it is not necessary to insist on the affection of the deep sensory fibers. The laboratory observations make the diagnosis of syphilis fairly certain, although the negative spinal fluid Wassermann tests, with the high cell count and the positive blood Wassermann reaction, is exceptional.

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Book Reviews

TOXICOMANIAS. By GREGORIO BERMANN. Pp. 382. Buenos Aires: Córdoba, 1926.

This book presents an interesting and well documented study on the psychologic, psychopathologic and social aspects of drug addiction, with special reference to conditions in the Argentine Republic, and, more especially, to the city of Córdoba. It is divided into seven parts, preceded by an introductory chapter. In the introduction, the author discusses the psychology of the drug appetite in the light of the opinions expressed by Legrain, and concludes that rather than as narcotic, such drugs as cocaine, morphine, opium, hasheesh, and even coffee and alcoholic beverages, act as euphoric agencies. Craving for these drugs and stimulants is the immediate desire for pleasure, not the desire for narcosis, which may at last result from large doses. This conclusion is reached after an analysis of the stimulation caused by different types of drugs.

In the first part the different types of drugs, including alcohol, and their effects are studied with some detail. The diffusion of the evil among civilized countries is also commented on. While several countries have tried to put the blame for the introduction of the drug habit on each other, Bermann wisely states that few students have faced the problem in a scientific and unprejudiced way, and still fewer are ready to face their country's shame. The causes of increased demand for drugs, according to Bermann, are: before the World War: (1) permanent desire for artificial stimulants; (2) desire of producers and their agents to increase their profits; (3) neuropathy and moral and mental weakness of persons and countries, a symptom of present-day decadence; (4) the social and economic condition of those who promote drug addiction. All of these causes were exaggerated during and after the war and in addition new causes arose, among which are: (1) the condition of collective neurosis and permanent insecurity; (2) loss of habits of discipline in work and lack of the latter after the war; (3) the uncontrollable desire for pleasure after the enforced temperance during the war, and the consequent corruption of social customs; (4) the large and illegitimate earnings during the war; (5) the wreck of ideals that formed the moral background of the combatants.

The last pages of the first part are devoted to a discussion of the question whether prohibition of alcoholic beverages is responsible for increase in the numbers of drug addicts. After an analysis of available (and much disputed) American statistics, and comments on the increase in the use of drugs in such countries as Argentina, where the traffic in alcohol has never been restricted, the author concludes that prohibition or restrictions on the use of alcoholic stimulants cannot be held responsible for increase in drug addiction.

The second part begins with a study of the social pathogeny of toxicomanias in Buenos Aires and in the rest of the Argentine, and a chapter is devoted to their origin and diffusion in Córdoba. From his observations in the latter city, covering a period of many years, the author reaches the conclusion that the habit is usually acquired through association with drug addicts in clubs, gangs and other places, largely as the result of a life of leisure among the higher classes, which seek new sensations and thrills. Many young persons acquired the habit in the belief that the use of drugs was fashionable and represented the last importation from highly civilized countries. A chapter is

devoted to the problem whether the drug habit must be regarded as a disease or a vice. In this respect, from his own experience, Bermann concludes that although there are cases of a clear neuropathic constitution among the addicts, there are just as many persons who would not necessarily fall prey to the desire for narcotics, but who contract the habit under the influence of social conditions.

The psychopathogeny of toxicomanias is examined in detail in the third part of the book. Here the author presents several cases of drug addiction of therapeutic origin and analyzes other cases which he labels as "toxicomanias of semi-medical cause." In the latter, physical pain is merely a circumstance, purely occasional, and acts as the first step in the initiation of the patient. In many cases such addicts have not been suffering from organic lesions for many years, and often one doubts whether the whole process was not psychic from the beginning. When morphinomaniacs do not clearly exhibit the physical cause of their reputed disease, the origin of drug craving is either psychopathologic or moral. The psychopathologic causes of toxicomanias are examined with detail in another chapter, in which the author discusses alcoholism and craving for alcohol ("alcoholimania"). The necessary condition for development of the latter is a certain state of mental and physical inferiority. If a subject, when in a depressed condition, drinks and afterward feels the euphoristic, stimulant and beneficial effects of alcohol, his necessity for a similar stimulation on other occasions will carry him to seek relief in alcoholic beverages. Periods of excessive drinking are often related to moral crises or difficulties of life or to psychophysical disorders that obey causes hitherto unknown. As soon as the euphoristic effect of alcohol ceases, the subjects are apt to feel more depressed and ill, and require more stimulants, the dose of which continuously increases according to the subject and his means. In extreme cases, alcoholic mania is as enslaving as a mania for alkaloids. In this respect alkaloids act in a somewhat similar way, and the effects sought in them are also euphoria, stimulation and elimination of painful sensations, whether moral or physical; at the same time, alkaloids lead to rest and mental relaxation, also to narcosis. But, while those who use alkaloids on account of pain obtain relief through avoidance of suffering, those in whom desire for drugs is a vice seek to find actual pleasure.

In the fourth part, devoted to constitution and psychopathogeny, the author discusses the importance of hereditary and constitutional factors predisposing to drug addition, and presents some family histories. His conclusions are that the percentage of abnormal persons in the families of toxicomaniac patients is higher than in normal families, and that deviations in the former are of wide range but seldom reach the state of insanity. In almost two thirds of the cases, alcoholism is a hereditary factor predisposing to drug addiction, and this factor is more manifest in morphinomaniac patients than in other types of addicts. The constitutional factor is expressed in neuropathy or psychopathy. Apparently, there is not a special predisposition for the formation of the drug habit, but merely the general predisposition present in abnormal persons. In addicts to cocaine there are all kinds of types of body, but among morphinomaniac patients, the asthenic and dysplastic types predominate. The problem of drug habit is also examined in the light of the theories of Wuth, and Joel and Frankel, and the author concludes that it is a physiologic process as well as psychic.

The psychopathology and criminology of drug addicts is examined in the fifth part of the book. Contrary to common opinion, Bermann believes that

it is difficult to give a general description of the features of drug addicts, and, in spite of his interest in the subject and his first-hand professional knowledge, he claims that in many cases it was impossible to detect any symptom of the habit. The dominant psychopathologic features are analyzed and the clinico-social types illustrated with concrete cases.

In the sixth part the forensic and penal aspects of the subject are discussed, more especially the wilful communication of the habit by one person to another. Cases of this type are illustrated with examples within the experience of the author. The book ends with considerations and suggestions on the suppression of the drug traffic and the treatment of drug addicts, and much constructive criticism is offered in this part.

DIE GRUNDLAGEN DER PSYCHOANALYSE. By HEINZ HARTMANN. Price, M. 8. Pp. 192. Leipzig: Georg Thieme, 1927.

To write an "Essentials" or "Principles" of psychoanalysis with its endless ramifications is no easy task, but the desirability of such a book is so obvious that it has repeatedly been attempted with varied degrees of success by many authors during the past twenty years, not only in Germany, but in France and in English speaking countries as well. As a matter of fact, the subject is so extensive that it cannot be well covered even in its essentials in any small volume today. However, Dr. Hartmann who is the assistant at the Psychiatric Clinic in Vienna, an institution for many years far from favorably inclined toward Freud's teachings, if not actually opposed to them, has accomplished his task with unusual skill. He has set for himself the double task of presenting the main features of psychoanalytic teachings and of investigating methodologically its foundations.

Under the term psychoanalysis he limits himself rigidly to that science which has been built up on the theoretical structure found in Freud's works and excludes the teachings of others, such as Jung, Adler and Steckel, whose inspiration undoubtedly came from psychoanalysis, but who have deviated from definite fundamental principles. A methodical critique of the theories of these offshoots of psychoanalysis would lead to different conclusions than that of the psychoanalytic method itself. Hartmann remarks that psychoanalysis is, to a greater extent than any other science, the work of one man, at least so far as its theoretical foundations are concerned, although both in its clinical and in its cultural applications others have contributed something of permanent value.

Having thus defined his task, Hartmann undertakes the consideration of psychoanalysis as a natural science, its relation to the cultural sciences, and finally the more commonly known precepts of the subject, such as the unconscious psychic dynamics, psychic energy (libido), with a chapter on psychoanalysis and psychiatry. Evidently under the influence of Schilder, the author has ever in mind the desirability of bringing into harmony pathology and psychophysical problems. Not only does Hartmann exhibit a thorough understanding of the psychoanalytic principles, but his book takes into consideration the most recent investigations of Freud, which appeared only last year in the monograph "Inhibition, Symptom and Anxiety." In writing this scholarly and thorough presentation which brings the subject in a condensed form and in its latest aspects to the reader, Hartmann has rendered a service never so well done before, although a possible objection may be that a considerable acquaintance with many of the phases of psychoanalysis which he covers is necessary for a ready understanding of this work.

THE ESCAPE FROM THE PRIMITIVE. By HORACE CARNCROSS, M.D., with a preface by SMITH ELY JELLIFFE, M.D. Price, $2.50. Pp. 348. New York: Charles Scribner's Sons, 1926.

Civilized man has evolved from more primitive forms of life not only in his body structure but also in his behavior, the manner in which he reacts to his surroundings in the primeval struggle for self and race preservation. In this striking book, the author not only endeavors to outline the evolution of conscious reactions from reflex or instinctive responses, but also points out that man today is still dominated in part by phylogenous or, as the psychoanalyst calls them, archaic mechanisms. He stresses the importance of discarding the old fallacy that more primitive means more natural. "The savage who lives primitively is no more a part of nature than the civilized man." In the first part of the book, the author discusses effectively and sanely the questions of chance or purpose, determinism and teleology, instinct and intelligence, free will and fate, and human responsibility in the phenomena of evolution.

In the second part of the book the evolution of man's conceptions of himself and his relations with the world around him and his longings for personal satisfaction and the perpetuation of life are traced through the concepts of magic, mysticism and religion, all primitively dominated by emotion. Gradually, intelligence emerges and knowledge of facts and sequences begins to assume control; the science of today begins to replace the magic of the primitive. The final section, "Child Man," reviews the development of the personality of the individual from childhood through adolescence to adulthood. Throughout, emphasis is laid on the incompleteness of present evolution and the hopes for the future with progressive elimination of instinctive reaction and the substitution of knowledge for superstition.

The book follows the principles and most recent teachings of Freud, but in such manner that even the most rabid opponent of the tenets built on the foundations of psychoanalysis will find little to arouse his ire. The English is excellent and, for the most part, is sufficiently nontechnical for the comprehension of the average well-educated man. "The Escape from the Primitive" is not a text book, but a fascinating exposition, admirably conceived and carried out, of the soundest elements of modern views on psychobiology, and will provide the reader with much food for thought and will unquestionably lead to broader comprehensions of man and humanity.

CRIMINAL INTELLIGENCE. By CARL MURCHISON, PH.D., Professor of Psychology in Clark University. Price, $4. Pp. 291. Worcester, Mass.: Clark University, 1926.

In this volume the author has collected the statistical results of alpha tests applied to convicts in the states of Ohio, Illinois, Indiana, Maryland and New Jersey which he has published in various journals. The results of similar tests among draftees in the army are used for comparison. The analyses deal with white native-born, white foreign-born and negro men, and there are added some studies of women criminals. Relations with geographic distribution, types of crime, recidivism, occupation, age and other factors are considered separately. The general conclusion reached is that the criminal is superior to the draftee in regard to intelligence, which "for the practical purpose of this book is whatever is expressed quantitatively as measured by the alpha test."

The quotation in the last sentence is an indication of the conservatism of the author in dealing with his facts, which constitute a contribution to

criminology of great value. Bearing in mind the conservatism of the author, the final chapter, of three pages, comes as a surprise. In it Murchison expresses some extremely drastic views on penology; these must be founded on facts other than those that form the chief topic of the book—the results obtained with the alpha tests. No consideration is given to factors in behavior other than the qualities which these tests measure.

ANLEITUNG ZU PSYCHIATRISCHEN UNTERSUCHUNGEN. By DR. HANS SEELERT. Price, M. 5.40. Pp. 181. Leipzig: Georg Thieme, 1926.

This introduction to the psychiatric examination takes up, chiefly on a question and answer basis, the process of determining such matters as affect, consciousness and intelligence in detail. There is some comment as to the prognostic meaning of the different facts which might be turned up. For instance, it is mentioned that in depressions, the patient in early stages is apt to express ideas and delusions concerning the present; a little later the ideas concern the future, and in the deepest depression the ideas turn toward the past. The method in the mental examination is first to collect the spontaneous utterances of the patient and then to follow with questions which are first general and then more and more specific. It would have to be an intelligent patient who could answer all the questions which the author suggests.

Shorter chapters describe the search for symptoms in the neurologic and physical fields and for pathogenesis. A final characteristic chapter on case histories warns against jumping at conclusions and putting in unessentials, and of the need to be objective in descriptions and to vary the questions according to the person. Somewhat more novel is the suggestion that the way to develop one's critical faculties is to write case records properly.

COMPENDIUM OF REGIONAL DIAGNOSIS IN AFFECTIONS OF THE BRAIN AND SPINAL CORD. A CONCISE INTRODUCTION TO THE PRINCIPLES OF CLINICAL LOCALIZATION IN DISEASES AND INJURIES OF THE CENTRAL NERVOUS SYSTEM. By ROBERT BING, Professor in the University of Basle. Translated from the 6th German Edition by F. S. Arnold, B.A., M.B., B.Ch. (Oxon). Price, $6.00. Pp. 198. St. Louis: The C. V. Mosby Co., 1927.

Bing's compendium has long been known as the best small book on the subject. It has deservedly grown into the sixth edition. The fifth edition appeared in 1923, and because of the fact that there have been so few advances in the knowledge of the nervous system, the sixth, the present edition, differs from the fifth only in that there has been a thorough revision of the illustrations, new diagrams having been added, and here and there are some minor changes in the text. With the revisions that have been made, this book can be recommended to students and neurologists as the best small book on organic nervous diseases published.

ANATOMICAL, PHYLOGENETICAL AND CLINICAL STUDIES ON THE CENTRAL NERVOUS SYSTEM. By B. BROUWER. Price, $2.50. Pp. 63. Baltimore: Williams and Wilkins Co., 1927.

Prof. B. Brouwer, of the University of Amsterdam, delivered the Herter lectures in April, 1926. This small volume contains a reprint of these lectures which were on the following subjects: (1) Projection of the Retina in the Brain; (2) Pathology of Sensibility; (3) Significance of Phylogenetic Studies for the Neurologist.

Professor Brouwer's work is too well known to need an extensive criticism, for these lectures are on subjects with which he has been identified for many years.

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CONTENTS

PAGE

STUDIES IN STUTTERING: INTRODUCTION. SAMUEL T. ORTON, M.D., IOWA CITY 671

STUDIES IN STUTTERING: I. DYSINTEGRATION OF THE BREATHING MOVEMENTS DURING STUTTERING. LEE EDWARD TRAVIS, PH.D., IOWA CITY 673

UNILATERAL (UNASSOCIATED) INNERVATION OF THE OCULAR MUSCLES. WILLIAM G. SPILLER, M.D., PHILADELPHIA 691

CHRONIC SUBDURAL ACCUMULATIONS OF CEREBROSPINAL FLUID AFTER CRANIAL TRAUMA: REPORT OF A CASE. IRA COHEN, M.D., WITH INTRODUCTORY REMARKS BY CHARLES A. ELSBERG, M.D., NEW YORK 709

TUMORS OF THE PINEAL GLAND. KEENE O. HALDEMAN, M.D., ROCHESTER, MINN 724

HEMANGIOMA OF THE SPINAL CORD. CARL W. RAND, LOS ANGELES 755

PERONEAL FORM OF PROGRESSIVE MUSCULAR ATROPHY: A CLINICAL REPORT OF TWO FAMILIES. ADOLPH EISENBUD, M.D., AND MORRIS GROSSMAN, M.D., NEW YORK 766

BRITISH AMERICAN NEUROLOGIC MEETING: SOME IMPRESSIONS. SIDNEY I. SCHWAB, M.D., ST. LOUIS 779

CLINICAL AND OCCASIONAL NOTES:

A CASE OF MULTIPLE SCLEROSIS COMPLICATED BY CANCEROUS METASTASES TO THE SPINE. JAMES H. HUDDLESON, M.D., NEW YORK 789

OBITUARIES:

THOMAS WILLIAM SALMON, M.D. 792

VINCENT GILIBERTI, M.D. 796

NEWS AND COMMENT 797

ABSTRACTS FROM CURRENT LITERATURE 798

SOCIETY TRANSACTIONS:

PHILADELPHIA NEUROLOGICAL SOCIETY 832

NEW YORK NEUROLOGICAL SOCIETY 846

SOCIÉTÉ DE NEUROLOGIE 859

BOOK REVIEWS 862